In 1997, the EPA established the NAAQS for PM2.5 as 15 μg/ m3. This was lowered to 12 μg/m3 in 2012. This standard has been largely justified on the basis of secret science epidemiology. These regulations are very powerful and impose huge costs on American businesses. The PM2.5 NAAQS, has been used to justify several multi-billion-dollar rules, such as the EPA Clean Power Plan and the CARB Truck and Bus Regulation.

Although a significant effect from such extremely low levels is on its face highly implausible, the stringent EPA regulations are justified primarily by a claim of preventing premature deaths, assuming a value of $10 million per statistical life saved. The controversy over the issue was brought to general attention in 2002 by Professor Robert Phalen.2

Epidemiology of Fine Particulate Matter

The EPA claim that PM2.5 causes "premature deaths" is based on epidemiologic cohort studies purporting to show that the relative risk (RR) for total mortality is slightly greater than 1.0 in U.S. populations exposed to higher levels of PM2.5. No etiologic mechanism has been established, and there is no experimental evidence that inhalation of 1 g or 5 g of PM2.5 can cause death. Weakly positive RRs do not prove causality. Major difficulties include: (1) geographic and temporal variation in PM2.5 mortality risk; (2) exaggeration of actual human exposure by PM2.5 monitors, which measure ambient outdoor levels

far from the subjects; and (3) confounding variables such as co-pollutants. Moreover, the key study relied on by EPA, the American Cancer Society (ACS) 1982 Cancer Prevention Study (CPS II)3 is seriously flawed.

Reanalysis of the American Cancer Society Cancer Prevention Study II (ACS CPS II)

CPS II began in 1982 and is similar to the original CPS I, which began in 1959. The seminal paper published by Pope et al. in 19953 was so controversial that the Health Effects Institute (HEI) sought applications from teams consisting of two to four epidemiologists, statisticians, and airpollution exposure experts to conduct a reanalysis, including "sensitivity analyses to test the robustness of the original findings and interpretations to alternative analytic approaches."4 The HEI Reanalysis published in 2000 did not complete the mandated sensitivity analysis to assess the effect of alternate data.5 HEI published a report in 2009,6 which extended the mortality follow-up of the study from 1989 to 2000, but it did not incorporate the EPA Inhalable Particulate Network (IPN) PM2.5 data7,8 that I had called to the authors' attention in my 2005 paper.9

In 2016 I was able to obtain access to data in an original 1982-1988 version of CPS II. The data had been previously inaccessible since 1995 despite a congressional subpoena and repeated requests by different agencies. I am the only independent scientist who has gained access to the individual level data in both CPS I and CPS II. I was able to reproduce the same key results as Pope et al. by doing exactly what the authors did in 1995.3 However, their results were sensitive to the PM2.5 data that they used and to their particular analysis.

HEI did not follow its own mandate to conduct a comprehensive reanalysis. In particular, their sensitivity analysis was not done properly. Of the 13 teams that submitted reanalysis applications, HEI selected a 31-member team based in Canada, headed by statistician Daniel Krewski. It included a geographer, Michael Jerrett, and another statistician, Richard Burnett, but only had one epidemiologist, Yue Chen. Chen's degree was from Shanghai Medical University, and he was not a coauthor on either the 2000 HEI report5 or the 2009 HEI report.6 Thus, to reanalyze a major U.S. epidemiological study, HEI used a Canadian team that had essentially no epidemiologist.

An early clue to the existence of problems is seen in Figure 21 in the 2000 HEI Reanalysis Report.5 (Figure 1 in this article.) This map shows that in 50 cities across the U.S. the level of PM2.5 mortality risk varies. Higher risks were found mainly in the Rust Belt or the Ohio Valley, and levels were actually reasonably low

in California and throughout most of the western part of the U.S. Beginning in 2002, I asked the head of HEI, Daniel Greenbaum, and its principal scientist, Aaron Cohen, to send me the underlying data for that map. For 16 years, they have consistently refused to reveal this data to me.

Fine Particles and Mortality Risk
Figure 1. PM2.5 Levels and Mortality Risk in the U.S. [Reprinted from 2000 HEI Reanalysis Report,5 with permission.]

Thus, using the HEI PM2.5 data of Pope et al., 3 there is a statistically significant slight increase in RR of 1.082. That means that if the PM2.5 level increases by 10 μg/m3, the risk of dying goes up by about 8%. But, using the IPN PM2.5 data, the effect is nonsignificant, RR = 1.025 (95% CI, 0.990-1.061). Note that if one divides the U.S. into the Ohio Valley (Indiana, Kentucky, Ohio, Pennsylvania, and West Virginia) and the rest of the country, the RR is indistinguishable from 1.0, no matter what PM2.5 data is used. Only by combining the Ohio Valley, which has both a higher mortality risk and a higher level of PM2.5, with the rest of the country can HEI show a statistically significant effect.

My reanalysis10 has been published online since Mar 28, 2017, and so far its validity has not been challenged. The selection of data by HEI was also very interesting, as seen in Table 2. There were actually 11 counties in California that were part of the IPN network, and the HEI analyses omitted 7 of the 11 counties for reasons the authors have not explained. HEI had data from 50 different cities, and the only ones they included from California were Fresno, Los Angeles, San Francisco, and San Jose (in Santa Clara County). Two other counties that represent the extremes in PM2.5 levels are highlighted in the table. The Pope 1995 paper3 was based primarily on these extremes. HEI had Albuquerque, N.M., at 9 μg/

My analysis of the CPS II data revealed that the county of residence of subjects could be approximated based on the ACS Division and Unit numbers. The CPS II data were collected by about 70,000 researchers, including myself, who enrolled 1.2 million subjects in Fall 1982. I performed an analysis comparable to the HEI Reanalysis, as shown in Table 1. The PM2.5 data labeled IPN in the table was published in EPA reports from the Inhalable Particulate Network (IPN) by David Hinton et al. in 19847 and 1986.8 Because of the evasions that I have experienced in attempting to obtain information from HEI, I took a closer look at the 2000 HEI Reanalysis Report and found it actually contains the data that I used, although in a mislabeled and somewhat altered form. I have designated that data as HEIDC, which is labeled PM2.5 DC in the 2000 Report. This data was indirectly referred to in a couple of places in the 2000 HEI report, although it was not analyzed.

m3, as the lowest value, and Huntington, W.V., at 34.4 μg/m3, as the highest value. This is curious because the data that comes from the IPN network actually shows different high and low values. In fact, there is no measurement in the IPN for Huntington, W.V., but rather for Wheeling, W.V., listed in the IPN column. From the table, both the low and the high values are in California, both of which omitted from the HEI analysis. The low value is 10.6 μg/m3 in Santa Barbara County, and the high value is 42.0 μg/m3 in Riverside County. The PM2.5 DC data that I found in the 2000 HEI Report appendix table, labeled HEIDC by me, had more than 50 cities, but only five of the 63 total cities were from California. The IPN network as a whole has about 85 cities. These major inconsistencies need to be addressed by these investigators. And so far, there is nothing but silence. This is only one of the issues that must be addressed if the investigators want to maintain any credibility.

Table 1. Enstrom Analyses of ACS CPS II Data Using Three Sources of PM2.5 Data

Table 2. Comparison of Data on PM2.5 and Mortality from Enstrom and HEI9

Relationship between PM2.5 and Mortality in California

Because of the Feb 26, 2010, conference in Sacramento, which I attended along with Professor Robert Phalen, other prominent scientists, and impacted business groups, we were able to get an analysis done by HEI that dealt with the California portion of the national CPS II results. The California data was partitioned out from the national analysis in the 2009 HEI Report.6 Based on the four HEI California counties shown in Table 2, the RR is about 0.9, significantly below 1.0, as shown in Table 3. This inverse relationship was reproduced using either the HEI data or the IPN data. Of course, this relationship cannot be etiologically correct, but it shows what can result from data omission and manipulation.

Table 3. Relative Risk for PM2.5 and Mortality in California Based on Four Counties

Table 4. PM2.5 and Total Mortality in Six California Cohorts Both my analysis and that by Thurston et al. on the NIH
AARP cohort,14 summarized in Table 5, show no effect nation- wide or in California.

There are actually six California cohorts that have been used to analyze the relationship between PM2.5 and total mortality, as shown in Table 4. The cohort that I initially used is labeled CA CPS I;9 the cohort used by Jerrett et al.11 is labeled CA CPS II. The Adventist Health Study of Smog (AHSMOG) was the original cohort study in California.12 There are also the California Teachers Cohort,10 the "West" portion of the Medicare Cohort Air Pollution Study (MCAPS),13 and the National Institutes of Health-American Association of Retired Persons (NIH AARP) cohort, which was published in 2016 by Thurston et al.14 The NIH AARP cohort is supposed to be an open access database, but is apparently currently controlled by Thurston. I have been able to get access to only the California portion of the data, and my analysis shows no effect in California. Averaging all six cohorts gives an RR of exactly 1.00, which means no relationship between PM2.5 and total mortality.
The lack of an effect in California might explain why Pope et al.3 omitted seven California cities from the national analysis. As Figure 1 shows, there is tremendous variation across the country. Yet the most severe regulations are in California, despite the clear absence of mortality risk there!

Table 5. Comparison of Enstrom and Thurston Analyses for
U.S. and California

An International Perspective on PM2.5

Despite the null effect shown by their own data and analyses, prominent advocates of drastic measures to reduce PM2.5 levels state in a major paper in the May 13, 2017, Lancet that ambient PM2.5 was the fifth-ranking mortality risk factor worldwide in 2015. Aaron J. Cohen, until recently HEI Principal Scientist, is the lead author, and Pope is a coauthor. The study is part of the World Health Organization (WHO) Global Burden of Disease (GBD) Project and was largely funded by HEI. The article claims that PM2.5 causes 4.2 million deaths annually worldwide, with 88,000 deaths in the U.S. (see Table 6). The mean PM2.5 level is

8.4 μg/m3 in the U.S. and 58.4 μg/m3 in China. Clearly, the PM2.5 level and premature deaths are low in the U.S. and high in China, India, and Africa.

Table 6. Global Deaths Attributed to PM 15

Agenda-driven Science

Since publishing my 2005 critique of the relationship between PM2.5 and total mortality9 and my 2017 critique,10 I have sent numerous requests to Pope, ACS, HEI, and others, inviting a rebuttal. I have received no response that confirms or refutes any of my analyses. It has, however, been incorrectly asserted that, "The study by Enstrom does not contribute to the larger body of evidence on the health effects of PM2.5." ACS has criticized me for having CPS II data that they have deliberately tried to keep secret. My invitations to authors and ACS officials to attend meetings, teleconferences, and symposia have simply been ignored. They even ignored an August 1, 2013, subpoena from the U.S. House Science, Space, and Technology Committee.

The control over air pollution research and assessments that is recognized by EPA is not based on special expertise in epidemiology. Pope, the self-proclaimed "world's leading expert on the effects of air pollution on health," is a professor of economics at Brigham Young University and holds a 1981 Ph.D. in agricultural economics from Iowa State University, where he studied the dynamics of crop yields. Michael Jerrett, who is one of the most prolific publishers and a member of the HEI reanalysis team, has a 1996 Ph.D. in geography from the University of Toronto, and no formal training in epidemiology. Aaron J. Cohen, until recently HEI's Principal Scientist, does hold a 1991 D.Sc. degree in epidemiology from Boston University, but he has badly misused the principles and standards of epidemiology. Although he supervised the 1998-2000 HEI Reanalysis Project, he has refused to clarify findings from this project and has refused to confirm or refute the findings in my 2017 CPS II reanalysis. It is very disturbing that ACS has allowed CPS II data to be used for more than 20 years for research that misuses the principles and standards of epidemiology and that has nothing significant to do with cancer.

The principal qualification for admission to the elite circle of influence appears to be dedication to the agenda of global controls on economic activity via air pollution regulations. The conclusion reached by researchers is apparently predetermined, as stated in the last paragraph of the GBD study on ambient air pollution: "As the experience in the U.S. suggests, changes in ambient PM2.5 associated with aggressive air quality management programmes, focused on major sources of air pollution including coal combustion, household burning of solid fuels, and road transport, can lead to increased life expectancy over short timeframes."15

What is the state of scientific integrity? It is very dangerous to one's career to criticize views backed by powerful interests, and I do it only because I believe current trends are anti- science and dangerous to our country. Simply being a passive observer is no longer acceptable.

To disclose my own background, I obtained a Ph.D. in physics in 1970, but I became an epidemiologist starting in 1973 in order to apply the rigorous principles of physics to observational epidemiology. I had a long career as a research professor and researcher at the UCLA School of Public Health. My research has examined the influence of environmental and lifestyle factors on mortality, and has on occasion reached politically incorrect conclusions. My research in air pollution epidemiology has been strongly influenced by Dr. Frederick Lipfert and Professor Robert Phalen. In February 2010 I was terminated from UCLA without warning and told that my "research is not aligned with the academic mission of the Department." In

February 2015 I settled a three-year federal whistleblower retaliation lawsuit against UCLA and my termination was reversed. My case and some of the issues related to my air pollution epidemiology research have been discussed in this journal.16

My background and publications, including rejections of my research, often without peer review, are documented on my website, www.scientificintegrityinstitute.org. I believe that major journals simply will not accept articles that challenge the established view. Moreover, authors of the papers promoting PM2.5 premature deaths omit null results, even their own. For example, Jerrett is the lead author of a 2007 study that shows no increased mortality associated with PM2.5 in the CPS II cohort if the results are divided into five time periods.17 Although researchers are paid millions of dollars, they're not under any obligation to address any of the concerns about their work. Those who disagree with the agenda are denied research funding.

We must prevent American science from following historical examples like that of Trofim Denisovich Lysenko. He was a phony plant geneticist, who gained the favor of Joseph Stalin because he didn't believe in Mendelian genetics. Lysenko's views controlled much of Soviet agriculture in the 1930s, 1940s, and 1950s, with devastating effect. False crop statistics were published, and dissenting scientists were purged. Nikolai Vavilov, a renowned plant geneticist, was imprisoned by Stalin and died of malnutrition.
Concerns about integrity in Western science are being raised. Richard Horton, editor of The Lancet, writes: "The case against science is straightforward: much of the scientific literature, perhaps half, may simply be untrue. Afflicted by studies with small sample sizes, tiny effects, invalid exploratory analyses, and flagrant conflicts of interest, together with an obsession for pursuing fashionable trends of dubious importance, science has taken a turn towards darkness."18

A U.S. House of Representatives bill called the Secret Science Reform Act was passed in 2014 and 2015 in order "to prohibit the Environmental Protection Agency from proposing, finalizing, and disseminating regulations or assessments based upon science that is not transparent or reproducible." The bill was revived in 2017 as the Honest and Open New EPA Science Treatment (HONEST) Act, labeled H.R. 1430, and was passed by the U.S. House of Representatives.
American science needs to guard against the heirs of Sinclair Lewis's protagonist in his 1927 novel Elmer Gantry, an itinerant preacher who is able to sell false religion to gullible people. We have prominent scientists who have successfully sold the notion that inhaling 1 g of invisible particles over an 80-year lifetime can cause premature death.

Conclusions

There is strong evidence from two large national cohorts that PM2.5 does not cause premature deaths in the US. There is strong evidence that this relationship has been falsified by EPA, the Health Effects Institute, and leading researchers for more than 20 years. Better oversight to assure scientific integrity, such as access to data, transparency, and consideration of opposing views, is imperative.

James E. Enstrom, Ph.D., M.P.H., a physicist and epidemiologist, is a retired research professor from the University of California, Los Angeles, and president of the Scientific Integrity Institute in Los Angeles. Contact: jenstrom@ucla.edu

REFERENCES

1. Enstrom JE. Scientific Misconduct in PM2.5 Epidemiology. Presented at 35th annual meeting, Doctors for Disaster Preparedness, New Orleans, La.; Aug 12, 2017, Available at: https://www.youtube.com/ watch?v=DaFUhJxMNco. Accessed Dec 26, 2017.
2. Phalen RF. The Particulate Air Pollution Controversy: ACase Studyand Lessons Learned. Dordrecht, The Netherlands: Kluwer Academic Publishers; 2002. Available at: http://www.amazon.com/gp/reader/1402072252/ref=si3_ rdr_ty. Accessed Feb 7, 2018.
3. Pope CA III, Thun MJ, Namboodiri MM. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Resp Crit Care Med 1995;151(3 pt 1):669–674. doi:10.1164/ajrccm.151.3.7881654.
4. HEI. A Request for Qualifications: Epidemiologists and Statisticians to Participate in a Reanalysis of Cohort Studies of Long-term Mortality and Particulate Air Pollution. Cambridge, Mass.: Heath Effects Institute; Jul 25, 1997. Available at: http://scientificintegrityinstitute.org/HEIRFQ072597. pdf. Accessed Dec 26, 2017.
5. Krewski D, Burnett RT, Goldberg MS, et al. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality: Special Report. Cambridge, Mass: Health Effects Institute; 2000. Available at: https://www.healtheffects.org/publication/ reanalysis-harvard-six-cities-study-and-american-cancer-society-study- particulate-air. Accessed Mar 18, 2018.

6. Krewski D, Jerrett M, Burnett RT. Extended follow-up and spatial analysis of the American Cancer Society Study linking particulate air pollution and mortality. HEI Research Report 140, Health Effects Institute, Boston, Mass.; 2009. Available at: https://www.healtheffects.org/publication/extended- follow-and-spatial-analysis-american-cancer-society-study-linking- particulate. Accessed February 20, 2017.
7. Hinton DO, Sune JM, Suggs JC, Barnard WF. Inhalable Particulate Network Report: Operation and Data Summary (Mass Concentrations Only). Volume I. April 1979-December 1982. EPA-600/4-84-088a. Research Triangle Park, N.C.: U.S. Environmental Protection Agency; November 1984. Available at: https://nepis.epa.gov/Exe/ZyPDF.cgi/20015OU3. PDF?Dockey=20015OU3.PDF. Accessed Feb 18, 2018.
8. Hinton DO, Sune JM, Suggs JC, Barnard WF. Inhalable Particulate Network Report: Data Summary (Mass Concentrations Only). Volume III. January 1983-December 1984. EPA-600/4- 86/019. Research Triangle Park, N.C.:
U.S. Environmental Protection Agency; April 1986. Available at: https:// nepis.epa.gov/Exe/ZyPDF.cgi/9101R4L8.PDF?Dockey=9101R4L8.PDF. Accessed Feb 18, 2018.
9. Enstrom JE. Fine particulate air pollution and total mortality among elderly Californians, 1973-2002. Inhal Toxicol 2005;17(14):803-816. PMID:16282158. http://scientificintegrityinstitute.org/IT121505.pdf. Accessed Dec 26, 2017.
10. Enstrom JE. Fine particulate matter and total mortality in cancer prevention study cohort reanalysis. Dose-Response 2017;1-12. doi: 10.1177/1559325817693345. Available at: http://journals.sagepub.com/ doi/pdf/10.1177/1559325817693345. Accessed Dec 26, 2017.
11. Jerrett M, Burnett RT, Pope CA III, et al. Spatiotemporal Analysis of Air Pollution and Mortality in California Based on the American Cancer Society Cohort. Revised Final Report for Contract No. 06-332 to CARB Research Screening Committee; Oct 28, 2011. Available at: http://www. arb.ca.gov/research/rsc/10-28-11/item1dfr06-332.pdf. Accessed Dec 26, 2017.
12. McDonnell WF, Nishino-Ishikawa N, Petersen FF, Chen LH, Abbey DE. Relationships of mortality with the fine and coarse fractions of long-term ambient PM10 concentrations in nonsmokers. JExpo Anal Environ Epidemiol 2000;10(5):427-436. Available at: http://www.scientificintegrityinstitute. org/JEAEE090100.pdf. Accessed Dec 26, 2017.

13. Zeger SL, Dominici F, McDermott A, Samet JM. Mortality in the Medicare population and chronic exposure to fine particulate air pollution in urban centers (2000-2005). Environ Health Perspect 2008;116:1614-1619.
14. Thurston GD, Ahn J, Cromar KR, et al. Ambient particulate matter air pollution exposure and mortality in the NIH-AARP Diet and Health Cohort. Environ Health Perspect 2016;124(4):484-490.
15. Cohen AJ, Burnett R, Anderson HR, et al. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015. Lancet 2017;389:1907-1918. Available at: https://pdfs.semanticscholar.org/a7f8/aa8d704c8c1c87d6a356de7e027828b529b4.pdf. Accessed Dec 27, 2017.
16. Arnett JC Jr. Politicized science: the case of Dr. James Enstrom v. powerful environmental activists. J Am Phys Surg 2012;17:118-119. Available at: http://www.jpands.org/vol17no4/arnett.pdf. Accessed Dec 27, 2017.
17. Jerrett M, Newbold KB, Burnett RT, et al. Geographies of uncertainty in the health benefits of air quality improvements. Stoch Environ Res Risk Assess 2007;21:511-522.
18. Horton R. Offline: What is medicine's 5 sigma? Lancet 2015;385:1380. Available at: http://www.thelancet.com/pdfs/journals/lancet/PIIS0140- 6736%2815%2960696-1.pdf. Accessed Dec 27, 2017

10. Dunn on US EPA Linear No Threshold Misconduct 2018.

This is a paper by the submitter Dunn that is intended to be an abstract as a presentation to a conference of the American Nuclear Society and the Health Physics Society on the problem of Linear No Threshold toxicology. The Conference is scheduled for early October 2018.

AN ENVIRONMENTAL NOBLE LIE,
LINEAR NO-THRESHOLD Radiation Biophysics Toxicology,
IT NEEDS TO GO

John Dale Dunn MD JD
American Nuclear Society/Health Physics Society Conference
Sept 30-Oct 3, 2018
Pasco, Washington

Abstract
The United States Environmental Protection Agency (USEPA) is charged with identifying and mitigating environmental risks. This article will discuss US EPA misguided decision to use Linear No Threshold as the template for Radiation Biophysics and Toxicology.
The Health Physics Society (HPS) has stated that reliance on the LNT model "...tends to foment the public's fear of all types of radiation . . . reliance on the LNT model, especially at very low doses and dose rates, is inappropriate and can exaggerate the risk." (Kirner 2017) (Ring et al. 2017). The HPS also condemns "collective" (cumulative) dose as a measure of biological radiation risk.
One hit or linear no threshold (LNT) radiation biophysics makes no sense as a theory for carcinogenesis. Most cancer cell types are hyper/multiploid due to telomeric mitotic dysfunction, not mutations of genetic code. Carcinogenesis is also enabled by immune system failure to eliminate malignant cell lines. Both phenomena are associated with aging.

The US EPA acceptance of the assertions on LNT of Biological Effects of Atomic Radiation (BEAR), Biological Effects of Ionizing Radiation (BEIR) and National Academy of Science (NAS) committees, has been so irrational as to assume there is no safe level of ionizing radiation. Nonsense.
The LNT cancer theorists ignore protective biological processes, even hormetic, certainly no effect evidence of low level radiation. (Ulsh 2010; Sacks and Siegel 2017; Welsh et al. 2017), Scott 2017), acknowledged by the National Council on Radiation Protection and Measurements (NCRP) over 15 years ago (NCRP 2001). "These experimental observations are not compatible with a single hit mechanism. . . hypothesis." (Trott and Rosemann 2000)
The fruit fly research by Hermann Muller and Curt Stern founded the LNT model, but the research actually showed a threshold, misrepresented by Muller, a committed advocate of LNT (Siegel et al. 2015; Calabrese 2017a, 2017b). Muller was a deceitful, relentless advocate of LNT, and, as a Nobel Laureate, very influential. (Calabrese 2017c)
The American Association of Physicists in Medicine (AAPM) strongly objects to the LNT approach as creating harm from adverse attitudes about imaging procedures. They consider the risks at or below 50 mSv [5 rem] for single procedures or 100 mSv [10 rem] for multiple procedures not detectable.
The USEPA use of LNT causes harm with no evidence of worthwhile benefit. US EPA claims that LNT is "conservative" and "cautious," translated as adoption of the misbegotten precautionary principle. The Fukishima mitigation, for example, was excessive, harmful and expensive, applied at doses far below the range of any negative public health consequences (Siegel et al. 2017c; Welsh et al. 2017).

Conclusions
The US EPA has been irresponsible and unscientific in its application of the Linear No Threshold template for radiation biophysics and toxicology. US EPA risk management is unscientific, unreliable and unjustified, wrongly derived from high dose rate environments and bench experimentation. Rat and mouse studies with exposures at lethal levels have created a long list of "carcinogens" that are then part of the LNT toxicology deception. (Calabrese 2018)
Society has become so fearful of radiation and chemicals that unnecessary steps are taken, and other risks are accepted, compliance costs are tolerated and are pursued energetically and expensively in a risk management environment of zero tolerance.
From the 1979 Three Mile Island to Fukishima in 2011, radiation incidents impacting large areas repeatedly show potential, variable risk for the immediate plant area, but, for example, even the terrible Chernobyl explosion, a stunningly limited harm from radiation beyond that.
The Fukishima event caused no radiation-related deaths (UNSCEAR 2013b), however the scare and the evacuation increased mortality, particularly in the elderly (Nomura et al. 2013; Yasumura et al. 2013; Uchimura et al. 2014, Ichiseki 2013) and the evacuations were scientifically unethical as a risk management strategy (Akabayashi and Hayashi 2012).
Changes, long overdue, on the matter of LDDR radiation risk management must go forward with the knowledge that adverse health effects are not detectable and that radiation exposures have a no effect, a harmful threshold of effect and even a sweet spot where radiation produces hormetic beneficial effects. (Calabrese 2013, Scott, 2017)
The USEPA Scientific Advisory Board (SAB) properly recommended a "change in the agency culture, change in how the agency works, and increased support for scientists and managers in programs and regional offices responsible for science integration." (Swackhamer and Burke 2012)
The radiation biophysics and toxicological precautionary principle needs a retirement in favor of rational risk assessment and mitigation.

References

Akabayashi A and Hayashi Y. 2012. Mandatory evacuation of residents during the Fukushima nuclear disaster: an ethical analysis. J Public Health (Oxf) 34:348-351

Calabrese E, Iavcoli I, Calbrese V, Hormesis: Its impact on medicine and health. Hum Exp Toxicol 2013 32: 120.-152.
http://het.sagepub.com/content/32/2/120
DOI: 10.1177/0960327112455069

Calabrese E. 2017a. The threshold vs LNT showdown: Dose rate findings exposed flaws in the LNT model Part 1. The Russell-Muller debate. Environ Res

Calabrese E. 2017b. The threshold vs LNT showdown: Dose rate findings exposed flaws in the LNT model part 2. How a mistake led BEIR I to adopt LNT. Environ Res

Calabrese E. 2017c. Obituary notice: LNT dead at 89 years, a life in the spotlight. Environmental Research 155 (2017) 276–278.

Calabrese E. 2018 From Muller to mechanism: How LNT became the default model for cancer risk assessment. Environmental Pollution 241 (2018) 289e302

Ichiseki H. 2013. Features of disaster-related deaths after the Great East Japan Earthquake. Lancet 381:204-204

Kirner NP. 2017. EPA Request for Regulatory Reform Task Force. McLean, VA

NCRP. 2001. Evaluation of the linear-nonthreshold dose-response model for ionizing radiation. 7910 Woodmont Avenue, Suite 800, Bethesda, MD 30814

NCRP. 2015. Health effects of low doses of radiation: Perspectives on integrating radiation biology and epidemiology. Bethesda, MD

Nomura S, Gilmour S, Tsubokura M, Yoneoka D, Sugimoto A, Oikawa T, Kami M and Shibuya K. 2013. Mortality risk amongst nursing home residents evacuated after the Fukushima nuclear accident: a retrospective cohort study. PLOS One 8:e60192

Ring JP, Tupin EA, Elder D, Hiatt J, Sheetz MA, Kirner NP and Little C. 2017. Health Physics Society comments to EPA Regulatory Reform Task Force. Health Phys in press:

Sacks B and Siegel JA. 2017. Preserving the anti-scientific linear no-threshold myth: Authority, agnosticism, transparency, and the standard of care. Dose Response 15:1559325817717839

Scott B, Small Radiation Doses Enhance Natural Barriers to Cancer. JPANDS 22:105-110. Winter 2017.

Siegel JA, Pennington CW, Sacks B and Welsh JS. 2015. The birth of the illegitimate linear no-threshold model: an invalid paradigm for estimating risk following low-dose radiation exposure. Am J Clin Oncol

Swackhamer DL and Burke TA. 2012. Personal communication with Jackson LP.

Trott KR and Rosemann M. 2000. Molecular mechanisms of radiation carcinogenesis and the linear, non-threshold dose response model of radiation risk estimation. Radiation and Environmental Biophysics 39:79-87

Uchimura M, Kizuki M, Takano T, Morita A and Seino K. 2014. Impact of the 2011 Great East Japan Earthquake on community health: ecological time series on transient increase in indirect mortality and recovery of health and long-term-care system. J Epidemiol Community Health 68:874-882

Ulsh BA. 2010. Checking the foundation: recent radiobiology and the linear no-threshold theory. Health Phys 99:747-758.

UNSCEAR. 2013b. Report to the General Assembly with Scientific Annexes: Volume I. New York, NY

Welsh JS, Sacks B and Siegel JA. 2017. Time to eliminate LNT: The NRC needs to adopt LT and eliminate ALARA. Nucl Med Biomed Imaging 2:1-5

Yasumura S, Goto A, Yamazaki S and Reich MR. 2013. Excess mortality among relocated institutionalized elderly after the Fukushima nuclear disaster. Public Health 127:186-188

Below is my abstract/monograph for a presentation to the Gulf Coast Geophysical Societies conference scheduled for late September of 2018. Here I summarize much of the research on human health impacts from warmer temperatures—that shows the benefits of warming. That debunks the catastrophic and ominous claims of the US EPA. There are certainly other reasons to object to US EPA clams that CO 2 is a pollutant and dangerous, but underlying those claims is their fraudulent and unsupported claim that warming would be deleterious to human health—when the opposite is true.

This if offered as just one exhibit that shows the US EPA has been irresponsible in its claims about the impact of CO2 rise and warming—there are other scientific research studies that show the claims about

11. Dunn on Global Warming and Climate Change EPA misconduct—the scam of making Carbon Dioxide a pollutant.

Warming is a Benefit to Humans and the Biosphere

John Dale Dunn MD JD

The Intergovernmental Panel on Climate Change (IPCC) predicts a global temperature increase of 3C or more by 2100, but other experts believe the best guess is 1C or less. We assert that increases in average temperature of the planet from the current 60 degrees F. will be beneficial to human health and the biosphere.

IPCC's alarms have led to widespread fear of the health effects of global warming (Schulte, 2008) and even political attack ads claiming people are dying of "carbon pollution" (WMC, 2015). These statements have no basis in scientific research and in fact and based on the evidence, warming will be a benefit to all living things. Carbon Dioxide that increases to even 1000 PPM will be beneficial to the biosphere and make the planet more hospitable and arable.

In fact, the litany of climate extremes postulated by the IPCC has been falsified by the actual record of climate measurements and observations. None of the environmental disasters, human displacements and disruptions predicted have come to pass during the past ten years, even as atmospheric carbon dioxide has continued to increase. We all know of the temperature "pause" that has accompanied an increase in atmospheric Carbon Dioxide.

In this document the benefits of fossil fuel use, and even warming, if it did occur, are explained in greater detail.

A warmer planet is beneficial to humanity as warmer temperatures lead to decreases in temperature-related mortality, premature deaths due to cardiovascular and respiratory disease, and stroke occurrences, and has little if any influence on vector-borne diseases such as malaria and dengue fever since vectors generally are not respectful of the definition of "tropical diseases.

Cool and colder temperatures kill while warmer temperatures are beneficial. It is troubling that, in the face of this evidence, environmentalists and politicians continue to frighten people with predictions of "killer heat waves" in a slightly warmer world. And yet, such claims are made. Severe heat waves are a weather phenomenon, not causally linked to average global temperature. Deaths from heat waves are most dramatic in areas with lack of adaptation—or general medical care for the disabled—who suffer from poor housing and medical problems that make them more susceptible.

References

Idso, C.D., Idso, S.B., Carter, R.M., and Singer, S.F. (Eds.) 2014. Climate Change Reconsidered II: Biological Impacts. Chicago, IL: The Heartland Institute.

IPCC. 2014. Summary for policymakers. In: Climate Change 2014: Impacts, Adaptation, and Vulnerability. PartA: Global and Sectoral Aspects. Contribution of Working Group II to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change [Field, C.B., V.R. Barros, D.J. Dokken,. J.Mach, M.D. Mastrandrea, T.E. Bilir, M. Chatterjee, K.L. Ebi, Y.O. Estrada, R.C. Genova, B. Girma, E.S. Kissel, A.N. Levy, S. MacCracken, P. R. Mastrandrea, and
L.L. White (Eds.)]. New York, NY: Cambridge University Press.
Schulte, K-M. 2008. Scientific consensus on climate change? Energy & Environment 19: 2. Tol, R.S.J. 2011. The economic impact of climate change in the 20th and 21st centuries.
Assessment Paper. Copenhagen Consensus on Human Challenges.
http://www.copenhagenconsensus.com/sites/default/files/climate_change.pdf. Last viewed on October 30, 2015.

Tol, R.S.J. 2013. Open letter to Professor Peter Høj, president and vice-chancellor, University of Queensland, August 2013. http://joannenova.com.au/2013/08/richard-tol-half-cooks-data-still- hidden-rest-shows-result-is-incorrect-invalid-unrepresentative/

Wisconsin Manufacturers and Commerce, 2015. Wisconsin chamber decries outrageous environmentalist ad attacking Sen. Johnson. News Release, September 3.

Global Warming and Mortality Rates

- Medical research confirms and explains why cooler, colder temperatures cause increased disease and death rates. Warmer temperatures are associated with health benefits and decreased deaths.

- Population studies around the world show that warmer temperatures lead to a net decrease in mortality worldwide, even in those areas described as tropical.

- Carbon dioxide (CO2) is invisible, odorless, nontoxic, and does not seriously affect human health until the CO2 content of the air reaches approximately 15,000 ppm, more than 37 times greater than the current concentration of atmospheric CO2 (Luft et al., 1974). There is no reason to be concerned about any direct adverse human health consequences of the ongoing rise in the air's CO2 content now or in the future,

currently at about 400 parts per million (0.04%) since even extreme model predictions by warming advocates are for less than 2000 parts per million (2%).

The Intergovernmental Panel on Climate Change (IPCC), however, sees looming health threats. The Summary for Policymakers of IPCC's Working Group II's report for the Fifth Assessment Report (AR5) identified eight "key risk factors" regarding the effect of climate change on human wellbeing, all of them allegedly "identified with high confidence" (IPCC, 2014, emphasis in original). They are:

i) Risk of death, injury, ill-health, or disrupted livelihoods in low-lying coastal zones and small island developing states and other small islands, due to storm surges, coastal flooding, and sea level rise. 37[RFC1-5]

ii) Risk of severe ill-health and disrupted livelihoods for large urban populations due to inland flooding in some regions. 38 [RFC 2 and 3]

iii) Systemic risks due to extreme weather events leading to breakdown of infrastructure networks and critical services such as electricity, water supply, and health and emergency services. 39 [RFC 2-4]

iv) Risk of mortality and morbidity during periods of extreme heat, particularly for vulnerable urban populations and those working outdoors in urban or rural areas. 40 [RFC 2 and 3]

v) Risk of food insecurity and the breakdown of food systems linked to warming, drought, flooding, and precipitation variability and extremes, particularly for poorer populations in urban and rural settings. 41 [RFC 2-4]

vi) Risk of loss of rural livelihoods and income due to insufficient access to drinking and irrigation water and reduced agricultural productivity, particularly for farmers and pastoralists with minimal capital in semi-arid regions. 42 [RFC 2 and 3]

vii) Risk of loss of marine and coastal ecosystems, biodiversity, and the ecosystem goods, functions, and services they provide for coastal livelihoods, especially for fishing communities in the tropics and the Arctic. 43 [RFC 1, 2, and 4]

viii) Risk of loss of terrestrial and inland water ecosystems, biodiversity, and the ecosystem goods, functions, and services they provide for livelihoods. 44 [RFC 1, 3, and 4]

There is no scientific basis for believing global temperatures will rise to levels high enough to bring about any of these risks. Indeed, there is sound scientific support for believing warming will be a net positive rather than negative.

Here, we summarize only research on the effects of rising global temperatures on human health and the medical literature shows warmer temperatures and a smaller difference between daily high and low temperatures that results from some rising temperatures as occurred during the twentieth and early twenty-first centuries, reduce mortality rates (the subject of this section) as well as illness and mortality due to cardiovascular and respiratory disease and stroke occurrence.

Similarly, the research is quite clear that climate has exerted only a minimal influence on recent trends in vector-borne diseases such as malaria, dengue fever, and tick-borne diseases. Other factors, many of them related to economic and technological setbacks or progress and not to weather, are far more important in determining the transmission and presence of these "tropical" diseases that are not so tropical at all.

Warmer Temperature Impacts on Human Health

- Warmer temperatures lead to a decrease in temperature-related mortality, including deaths associated with cardiovascular disease, respiratory disease, and strokes. The evidence of this benefit comes from research conducted in every major country of the world.

- In the United States the average person who died because of cold temperature exposure lost in excess of 10 years of potential life, whereas the average person who died because of extreme heat related event lost no more than a few days or weeks of life because heat has a greater effect on more seriously debilitated and ill persons.

- In the U.S., some 4,600 deaths are delayed each year as people move from cold northeastern states to warm southwestern states. Between 3 and 7% of the gains in longevity experienced over the past three decades was due simply to people moving to warmer states.

- Cold-related deaths are far more numerous than heat-related deaths in the United States and the world. Coronary (heart attack) and cerebral thrombosis (stroke) account for about half of all cold-related mortality, events that are directed related to blood vessel and blood viscosity effects of cool or cold environments.

- Global warming, if it did occur, even to the degree predicted in the extreme, will reduce the incidence of cardiovascular diseases related to low temperatures and wintry weather by a much greater degree than the warming might increase the incidence of deaths or illness attributable to heat. Heat illness primarily produces fluid and electrolyte disturbances, loss of core temperature control and organ dysfunction from dehydration, circulatory failure and heat caused stress, not clotting events.

- The heat wave deaths of 1995 in Chicago and 2003 in Europe are pointed to by advocates of the claim that heat stress deaths will increase with any warming that might occur, but a closer look at heat event death rates in some of the studies below show acclimation increased awareness have blunted any heat stress death increases. In the case of Chicago and Europe temps rose to over 100 but the availability of air conditioning and ventilation along with attention to the needs of elderly and disabled individuals was determined to be a major reason for heat deaths.

- The heat deaths that occur during severe heat events are the result of stress and inability to acclimate to maintain normal core temperature control and avoid dehydration. Acclimatization and proper attention to the vulnerable populations failed in Chicago in 1995 and Europe, particularly France in 2003, for example with hundreds of heat deaths in the former and 20,000 or more deaths in the later.

- A large body of scientific examination and research contradicts and disproves the claim that malaria will expand across the globe and intensify as a result of CO2-induced warming. Malaria is historically a disease that was endemic to cool and even cold climates like Finland and Russia but has been suppressed by hygienic and vector control

measures.
- Concerns over large increases in vector-borne diseases such as dengue as a result of rising temperatures are unfounded and unsupported by the scientific literature, as climatic indices are poor predictors for dengue disease. The Aedes Aegypti Anopheles and Asian Tiger mosquitos all have been found at higher latitudes.
- While temperature and climate effect the geographical distribution of ticks, they are not among the significant factors determining the incidence of tick-borne diseases. Moreover the effect of small increases in climate temperature, if does occur with certainly not impact the range of ticks that now live in the high latitudes, even in the mountains of those high latitudes.

References

Idso, C.D., Idso, S.B., Carter R.M., and Singer, S.F. (Eds.) 2014. Climate Change Reconsidered II: Biological Impacts. Chicago, IL: The Heartland Institute

IPCC. 2014. Summary for policymakers. In: Climate Change 2014: Impacts, Adaptation, and Vulnerability. PartA: Global and Sectoral Aspects. Contribution of Working Group II to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change [Field, C.B., V.R. Barros, D.J. Dokken,. J.Mach, M.D. Mastrandrea, T.E. Bilir, M. Chatterjee, K.L. Ebi, Y.O. Estrada, R.C. Genova, B. Girma, E.S. Kissel, A.N. Levy, S. MacCracken, P. R. Mastrandrea, and
L.L. White (Eds.)]. New York, NY: Cambridge University Press.

Luft, U.C., Finkelstein, S., and Elliot, J.C. 1974. Respiratory gas exchange, acid-base balance, and electrolytes during and after maximal work breathing 15 mm Hg PICO2. In: Nahas, G. and Schaefer, K.E. (Eds.) Carbon Dioxide and Metabolic Regulations. New York, NY: Springer- Verlag. 273–281.

Basis in Medical Science

Medical science explains why colder temperatures often cause diseases and sometimes fatalities whereas warmer temperatures are associated with health benefits.

Wang et al. collected daily mortality and meteorological data from 66 communities across China over the period 2006-2011. They then subjected these data to a series of analyses to elucidate the relationship between cold spell characteristics and human mortality. And what did those analyses reveal?

Not surprisingly, cold spells significantly increased human mortality risk in China. As indicated in Figure 1 below, the combined cumulative excess mortality risk (CER) for all of China when defining cold spells with a 5th and 2.5th percentile temperature intensity threshold was 28.5 and 39.7 percent, respectively. However, there were notable geographic differences; CER was tempered and near zero in the colder/higher latitudes, but increased to 58.7 and 92.9 percent at the corresponding 5th and 2.5th percentile temperature intensity

thresholds for the warmest and most southern latitude. Such geographic differences in mortality risk, according to the authors, are likely the product of better physiological and behavioral acclimatization of the northerly populations to cold weather.

Clearly, cold spells kill; and as has been found in almost every study of the subject, the risk of death from cold spells far exceeds that from heat waves (see the many reviews we have posted on this topic confirming this fact in our Subject Index under the heading Health Effects of Temperature: Hot vs, Cold Weather). As such, therefore, a little global warming would likely result in a net saving of lives by reducing the number of deaths that occur at the cold end of the temperature spectrum.

Antonio Gasparinni (2015) was lead author for a large international group of researchers who studied the effect of temperature extremes on death rates. Gasparrini and his co-authors analyzed data from 384 locations including the countries of Australia, Brazil, Canada, China, Italy, Japan, South Korea, Spain, Sweden, Taiwan, Thailand, the United Kingdom and the United States of America. By fitting a standard time-series Poisson model to the data obtained for each location, while controlling for trends and day of the week, they estimated temperature-mortality associations with a distributed lag non-linear model with 21 days of lag, after which they pooled the results they obtained in a multivariate meta-regression that included country indicators and temperature averages and ranges.

This work allowed them to calculate the number of human deaths attributable to heat and cold -- defined as temperatures above and below the optimum (minimum mortality) temperature -- for both moderate and extreme temperatures, the latter being defined "using cutoffs at the 2.5th and 97.5th temperature percentiles." And what did they thereby learn?

Based on data pertaining to a total of 74,225,200 human deaths that occurred between 1985 and 2012, the 23 researchers determined that 7.71% of the lives lost were caused by non-optimum temperatures; and among this group they found that "more temperature-attributable deaths were caused by cold (7.29%) than by heat (0.42%)" which makes cold in excess of seventeen times more deadly than heat. And they add, in this regard, that moderate "hot and cold temperatures represented most of the total health burden." Consequently, it seems pretty clear that any successful attempt to reverse or slow any potential increase in Earth's mean global temperature would likely come at a net cost of many human lives the world over, not a savings. The Gasparrini research provides a compelling confirmation of the reality that warmer temperatures are better for human welfare than cooler or colder temperatures. (Gasparrini Lancet 2015)

Keating and Donaldson (2001) explain that "cold causes mortality mainly from arterial thrombosis and respiratory disease, attributable in turn to cold-induced hemoconcentration and hypertension [in the first case] and respiratory infections [in the second case]." McGregor (2005) notes "anomalous cold stress can increase blood viscosity and blood pressure due to the activation of the sympathetic nervous system which accelerates the heart rate and increases vascular resistance (Collins et al., 1985; Jehn et al., 2002; Healy, 2003; Keatinge et al., 1984; Mercer, 2003; Woodhouse et al., 1993)," adding, "anomalously cold winters may also increase other risk factors for heart disease such as blood clotting or fibrinogen concentration, red blood cell count per volume and plasma cholesterol."

Wang et al. (2013) write, "A large change in temperature within one day may cause a sudden change in the heart rate and circulation of elderly people, which all may act to increase the risk of cardiopulmonary and other diseases, even leading to fatal consequences." This is significant for the climate change debate because, as Wang et al. also observe, "it has been shown that a rise of the minimum temperature has occurred at a rate three times that of the maximum temperature during the twentieth century over most parts of the world, which has led to a decrease of the diurnal temperature range (Karl et al., 1984, 1991)."

Robeson (2002) demonstrated, based on a 50-year study of daily temperatures at more than 1,000 U.S.

weather stations that daily (diurnal) temperature variability declines with warming and at a very substantial rate, so this aspect of a warmer world would lead to a reduction in temperature- related deaths.
Clearly, cold spells kill; and as has been found in almost every study of the subject, the risk of death from cold spells far exceeds that from heat waves. As such, therefore, a little global warming would likely result in a net saving of lives by reducing the number of deaths that occur at the cold end of the temperature spectrum.

Keatinge and Donaldson (2004) report coronary and cerebral thrombosis account for about half of all cold-related deaths, and respiratory diseases account for approximately half of the rest. They say cold stress causes an increase in arterial thrombosis "because the blood becomes more concentrated, and so more liable to clot during exposure to cold." As they describe it, "the body's first adjustment to cold stress is to shut down blood flow to the skin to conserve body heat," which "produces an excess of blood in central parts of the body," and to correct for this effect, "salt and water are moved out from the blood into tissue spaces," leaving behind "increased levels of red cells, white cells, platelets and fibrinogen" that lead to increased viscosity of the blood and a greater risk of clotting.
Keatinge and Donaldson also note "cold spells are closely associated with sharp increases in mortality rates," and "deaths continue for many days after a cold spell ends." On the other hand, they report, "increased deaths during a few days of hot weather are followed by a lower than normal mortality rate," because "many of those dying in the heat are already seriously ill
and even without heat stress would have died within the next 2 or 3 weeks."
With respect to the implications of global warming for human mortality, Keatinge and Donaldson state "since heat-related deaths are generally much fewer than cold-related deaths, the overall effect of global warming on health can be expected to be a beneficial one." They report, "The rise in temperature of 3.6°F expected over the next 50 years would increase heat-related deaths in Britain by about 2,000 but reduce cold-related deaths by about 20,000."
Keating and Donaldson's reference to deaths that typically would have occurred shortly even without excess heat is a phenomenon researchers call "displacement" or "harvesting." A study from Germany found "cold spells lead to excess mortality to a relatively small degree, which lasts for weeks," while "the mortality increase during heat waves is more pronounced, but is followed by lower than average values in subsequent weeks" (Laschewski and Jendritzky, 2002). The authors say the latter observation suggests people who died from short-term exposure to heat possibly "would have died in the short term anyway." They found the mean duration of above-normal mortality for the 51 heat episodes that occurred from 1968 to 1997 was 10 days, with a mean increase in mortality of 3.9%, after which there was a mean decrease in mortality of 2.3% for 19 days. Hence, the net effect of the two perturbations was an overall decrease in mortality of 0.2% over the full 29-day period.
The US EPA web site discussion of heat wave deaths referenced below reveals that the EPA recognizes heat wave deaths are not reliably counted because of loose death certificate definitions of heat caused versus heat related. Cardiovascular deaths is used as a catch all descriptor Although the deaths attributed to severe heat waves are described as Cardiovascular, the mechanism is metabolic and physiologic dysfunction and a collapse of the systems that maintain temperature equilibrium in endotherms like humans. The victims don't die of a heart attack, a coronary ischemic event caused by clots and narrowed coronary arteries, an occlusive event, they die of temperature effects and the failure of internal systems, including lung and cardiovascular system, solid organ, and brain malfunctions in the face of heat stress, dehydration, and rising core temperatures, along with dehydration and loss of mechanisms to maintain normal temperature. The victims are debilitated, and live in a stressfully hot environment and succumb for failure to acclimate and maintain normal body physiology.

References

Collins, K.J., Easton, J.C., Belfield-Smith, H., Exton-Smith, A.N., and Pluck, R.A. 1985. Effects of age on body temperature and blood pressure in cold environments. Clinical Science 69: 465– 470.

Gasparrini A., Guo Y., Hashizume M., et.al.,. Mortality risk attributable to high and low ambient temperature: a multi-country observational study. The Lancet: 10.1016/S0140-6736(14)62114-0. 20 May 2015.

Healy, J.D. 2003. Excess winter mortality in Europe: a cross country analysis identifying risk factors. Journal of Epidemiology and Public Health 57: 784–789.

Jehn, M., Appel, L.J., Sacks, F.M., and Miller III, E.R. 2002. The effect of ambient temperature and barometric pressure on ambulatory blood pressure variability. American Journal of Hypertension 15: 941–945.

Karl, T.R., Jones, P.D., Knight, R.W., Kukla, G., Plummer, N., Razuvayev, V., Gallo, K.P., Lindseay, J., Charlson, R.J., and Peterson, T.C. 1984. A new perspective on recent global warming: asymmetric trends of daily maximum and minimum temperature. Bulletin of the American Meteorological Society 74: 1007–1023.

Karl, T.R., Kukla, G., Razuvayev, V.N., Changery, M.J., Quayle, R.G., Heim Jr., R.R., Easterling, D.R., and Fu, C.B. 1991. Global warming: evidence for asymmetric diurnal temperature change. Geophysical Research Letters 18: 2253–2256.

Keatinge, W.R. and Donaldson, G.C. 2004. The impact of global warming on health and mortality. Southern Medical Journal 97: 1093–1099.

Keatinge, W.R., Donaldson, G.C., Cordioli, E., Martinelli, M., Kunst, A.E., Mackenbach, J.P., Nayha, S., and Vuori, I. 2000a. Heat related mortality in warm and cold regions of Europe: Observational study. British Medical Journal 321: 670–673.

Laschewski, G. and Jendritzky, G. 2002. Effects of the thermal environment on human health: an investigation of 30 years of daily mortality data from SW Germany. Climate Research 21: 91– 103.

McGregor, G.R. 2005. Winter North Atlantic Oscillation, temperature and ischemic heart disease mortality in three English counties. International Journal of Biometeorology 49: 197–204.

Mercer, J.B. 2003. Cold—an underrated risk factor for health. Environmental Research 92: 8–13.

Robeson, S.M. 2002. Relationships between mean and standard deviation of air temperature: implications for global warming. Climate Research 22: 205–213.

Shreuder, C, "The 1995 Chicago Heat Wave" Chicago Tribune July 15, 2015
http://www.chicagotribune.com/news/nationworld/politics/chi-chicagodays-1995heat-story- story.html

UK Met Office "The Heat Wave of 2003"
http://www.metoffice.gov.uk/learning/learn-about-the-weather/weather-phenomena/case- studies/heatwave

US EPA Climate Change Indicators: Heat-Related Deaths. https://www.epa.gov/climate- indicators/climate-change-indicators-heat-related-deaths

Wang, M-z., Zheng, S., He, S-l., Li, B., Teng, H-j., Wang, S-g., Yin, L., Shang, K-z., and Li, T-s. 2013. The association between diurnal temperature range and emergency room admissions for cardiovascular, respiratory, digestive and genitourinary disease among the elderly: A time series study. Science of the Total Environment 456–457: 370–375.

Wang, L., Hu, M., Zeng, W., Zhang, Y., Rutherford, S., Lin, H., Xiao, J., Yin, P., Liu, J., Chu, C., Tong, S., Ma, W. and Zhou, M. 2016. The impact of cold spells on mortality and effect modification by cold spell characteristics. Scientific Reports 6: 38380, DOI: 10.1038/srep38380

Woodhouse, P.R., Khaw, K., and Plummer, M. 1993. Seasonal variation of blood pressure and its relationship to ambient temperature in an elderly population. Journal of Hypertension 11: 1267–1274.
Observational Research in Asia

Behar (2000) studied sudden cardiac death (SCD) and acute myocardial infarction (AMI) in Israel, concentrating on the role temperature may play in the incidence of these health problems. Behar notes "most of the recent papers on this topic have concluded that a peak of SCD, AMI and other cardiovascular conditions is usually observed in low temperature weather during winter." He cites an Israeli study by Green et al. (1994), which reported between 1976 and 1985 "mortality from cardio-vascular disease was higher by 50% in mid-winter than in mid-summer, both in men and women and in different age groups," even though summer temperatures in the Negev, where much of the work was conducted, often exceed 30°C and winter temperatures typically do not drop below 10°C. Behar concludes these results "are reassuring for populations living in hot countries."

Kan et al. (2003) investigated the association between temperature and daily in Shanghai, China, finding a V-like relationship between total mortality and temperature that had a minimum mortality risk at 26.7°C. Above this optimum temperature, they observe, "total mortality increased by 0.73% for each degree Celsius increase; while for temperatures below the optimum value, total mortality decreased by 1.21% for each degree Celsius increase." The net effect of a warming in Shanghai, China, therefore, would likely be reduced mortality on the order of 0.5% per degree Celsius increase in temperature, or perhaps more.

Guo et al. (2012) examine the nonlinear and delayed effects of temperature on cause- specific and age-specific mortality employing data from 1999 to 2008 for Chiang Mai, Thailand with a population of 1.6 million people. Controlling for season, humidity, ozone, and particulate matter (PM10) pollution, the three researchers found "both hot and cold temperatures resulted in immediate increase in all mortality types and age groups," but "the hot effects on all mortality types and age groups were short-term, while the cold effects lasted longer." The cold effects were greater, with more people dying from them than from the effects of heat.

Lindeboom et al. (2012) used daily mortality and weather data for the period 1983–2009 pertaining to Matlab, Bangladesh, to measure lagged effects of weather on mortality, controlling for time trends and

seasonal patterns. The four researchers report "mortality in the Matlab surveillance area shows overall weak associations with rainfall, and stronger negative association with temperature." They determined there was "a 1.4% increase in mortality with every 1°C decrease in mean temperature at temperatures below 29.2°C," but only "a 0.2% increase in mortality with every 1°C increase in mean temperature."

Wang et al. (2013) evaluated the short-term effect of diurnal temperature range (DTR) on emergency room (ER) admissions among elderly adults in Beijing. The nine researchers report "significant associations were found between DTR and four major causes of daily ER admissions among elderly adults in Beijing." They state "a 1°C increase in the 8-day moving average of DTR (lag 07) corresponded to an increase of 2.08% in respiratory ER admissions and 2.14% in digestive ER admissions," and "a 1°C increase in the 3-day and 6-day moving average of DTR (lag 02 and lag 05) corresponded to a 0.76% increase in cardiovascular ER admissions, and a 1.81% increase in genitourinary ER admissions, respectively.

Wu et al. (2013) assessed the health effects of temperature on mortality in four subtropical cities of China (Changsha, Kunming, Guangzhou, and Zhuhai). The 11 researchers report a U-shaped relationship between temperature and mortality was found in the four cities, indicating "mortality is usually lowest around a certain temperature and higher at lower or higher temperatures." Although "both low and high temperatures were associated with increased mortality in the four subtropical Chinese cities," Wu et al. state the "cold effect was more durable and pronounced than the hot effect."

References

Behar, S. 2000. Out-of-hospital death in Israel—Should we blame the weather? Israel Medical Association Journal 2: 56–57.

Cheng, Y. and Kan, H. 2012. Effect of the interaction between outdoor air pollution and extreme temperature on daily mortality in Shanghai, China. Journal of Epidemiology 22: 28–36.

Green, M.S., Harari, G., and Kristal-Boneh, E. 1994. Excess winter mortality from ischaemic heart disease and stroke during colder and warmer years in Israel. European Journal of Public Health 4: 3–11.

Guo, Y., Punnasiri, K., and Tong, S. 2012. Effects of temperature on mortality in Chiang Mai city, Thailand: a time series study. Environmental Health: http://ehjournal.net/content/11/1/36.

Kan, H., London, S.J., Chen, H., Song, G., Chen, G., Jiang, L., Zhao, N., Zhang, Y., and Chen, B. 2007. Diurnal temperature range and daily mortality in Shanghai, China. Environmental Research 103: 424–431.

Kan, H-D., Jia, J., and Chen, B-H. 2003. Temperature and daily mortality in Shanghai: A time- series study. Bio-medical and Environmental Sciences 16: 133–139.

Karl, T.R., Jones, P.D., Knight, R.W., Kukla, G., Plummer, N., Razuvayev, V., Gallo, K.P., Lindseay, J., Charlson, R.J., and Peterson, T.C. 1984. A new perspective on recent global warming: asymmetric trends of daily maximum and minimum temperature. Bulletin of the American Meteorological Society 74: 1007–1023.

Karl, T.R., Kukla, G., Razuvayev, V.N., Changery, M.J., Quayle, R.G., Heim Jr., R.R., Easterling, D.R., and Fu, C.B. 1991. Global warming: evidence for asymmetric diurnal temperature change. Geophysical Research Letters 18: 2253–2256.

Ma, W., Xu, X., Peng, L., and Kan, H. 2011. Impact of extreme temperature on hospital admission in Shanghai, China. Science of the Total Environment 409: 3634–3637.

Tan, J., Zheng, Y., Song, G., Kalkstein, L.S., Kalkstein, A.J., and Tang, X. 2007. Heat wave impacts on mortality in Shanghai, 1998 and 2003. International Journal of Biometeorology 51: 193–200.

Touloumi, G., Samoli, E., and Katsouyanni, K. 1996. Daily mortality and "winter type" air pollution in Athens, Greece—a time series analysis within the APHEA project. Journal of Epidemiology and Community Health 50: Supplement 1: 47–51.

Wang, M-z., Zheng, S., He, S-l., Li, B., Teng, H-j., Wang, S-g., Yin, L., Shang, K-z., and Li, T-s. 2013. The association between diurnal temperature range and emergency room admissions for cardiovascular, respiratory, digestive and genitourinary disease among the elderly: A time series study. Science of the Total Environment 456–457: 370–375.

Wong, C.M., Ma, S., Hedley, A.J., and Lam, T.H. 1999. Does ozone have any effect on daily

hospital admissions for circulatory diseases? Journal of Epidemiology and Community Health 53: 580–581.

Wong, C.M., Ma, S., Hedley, A.J., and Lam, T.H. 2001. Effect of air pollution on daily mortality in Hong Kong. Environmental Health Perspectives 109: 335–340.

Wu, W., Xiao, Y., Li, G., Zeng, W., Lin, H., Rutherford, S., Xu, Y., Luo, Y., Xu, X., Chu, C., and Ma, W. 2013. Temperature-mortality relationship in four subtropical Chinese cities: A time- series study using a distributed lag non-linear model. Science of the Total Environment 449: 355–362.

Yang, J., Liu, H.-Z., Ou, C.-Q., Lin, G.-Z., Zhou, Q., Shen, G.-C., Chen, P.-Y., and Guo, Y. 2013. Global climate change: Impact of diurnal temperature range on mortality in Guangzhou, China. 2013. Environmental Pollution 175: 131–136.

Zhang, Y., Huang, W., London, S.J., Song, G., Chen, G., Jiang, L., Zhao, N., Chen, B., and Kan, H. 2006. Ozone and daily mortality in Shanghai, China. Environmental Health Perspectives 114: 1227–1232.

Observational Research in Europe

Keatinge and Donaldson (2001) analyzed the effects on human mortality of temperature, wind, rain, humidity, and sunshine during high pollution days in the greater London area over the period 1976–1995. They observed simple plots of mortality rate versus daily air temperature revealed a linear increase as temperatures fell from 15°C to near 0°C. Mortality rates at temperatures above 15°C, however, were

"grossly alinear," as they describe it, showing no trend. Only low temperatures were found to have a significant effect on immediate and long-term mortality. They conclude "the large, delayed increase in mortality after low temperature is specifically associated with cold and is not due to associated patterns of wind, rain, humidity, sunshine, SO2, CO, or smoke."

Kysely and Huth (2004) calculated deviations of the observed number of deaths from the expected number of deaths for each day of the year in the Czech Republic for the period 1992– 2000. They found "the distribution of days with the highest excess mortality in a year is clearly bimodal, showing a main peak in late winter and a secondary one in summer." Regarding the smaller number of summer heat-wave-induced deaths, they also found "a large portion of the mortality increase is associated with the harvesting effect, which consists in short-term shifts in mortality and leads to a decline in the number of deaths after hot periods (e.g. Rooney et al., 1998; Braga et al., 2002; Laschewski and Jendritzky, 2002)." For the Czech Republic, they report, "the mortality displacement effect in the severe 1994 heat waves can be estimated to account for about 50% of the total number of victims." As they describe it, "people who would have died in the short term even in the absence of oppressive weather conditions made up about half of the total number of deaths."

Diaz et al. (2005) examined the effect of extreme winter temperature on mortality in Madrid, Spain for people older than 65, using data from 1,815 winter days over the period 1986– 1997, during which time 133,000 deaths occurred. They found that as maximum daily temperature dropped below 6°C, which they describe as an unusually cold day (UCD), "the impact on mortality also increased significantly." They also found the impact of UCDs increased as the winter progressed, with the first UCD of the season producing an average of 102 deaths/day at a lag of eight days and the sixth UCD producing an average of 123 deaths/day at a lag of eight days.

Laaidi et al. (2006) conducted an observational population study in six regions of France between 1991 and 1995 to assess the relationship between temperature and mortality in areas of widely varying climatic conditions and lifestyles. In all cases they found "more evidence was collected showing that cold weather was more deadly than hot weather." These findings, the researchers say, are "broadly consistent with those found in earlier studies conducted elsewhere in Europe (Kunst et al., 1993; Ballester et al., 1997; Eurowinter Group, 1997; Keatinge et al., 2000; Beniston, 2002; Muggeo and Vigotti, 2002), the United States (Curriero et al., 2002) and South America (Gouveia et al., 2003)." They also say their findings "give grounds for confidence in the near future," stating even a 2°C warming over the next half century "would not increase annual mortality rates."

Analitis et al. (2008) analyzed short-term effects of cold weather on mortality in 15 major European cities using data from 1990–2000, and found "a 1°C decrease in temperature was associated with a 1.35% increase in the daily number of total natural deaths and a 1.72%, 3.30% and 1.25% increase in cardiovascular, respiratory, and cerebro-vascular deaths, respectively." In addition, they report "the increase was greater for the older age groups," and the cold effect "persisted up to 23 days, with no evidence of mortality displacement." They conclude their results "add evidence that cold-related mortality is an important public health problem across Europe and should not be overlooked by public health authorities because of the recent focus on heat-wave episodes."

Wichmann et al. (2011) investigated the association between the daily three-hour maximum apparent temperature (which reflects the physiological experience of combined exposure to humidity and temperature) and deaths due to cardiovascular disease (CVD), cerebrovascular disease (CBD), and respiratory disease (RD) in Copenhagen over the period
1999–2006.

Monthly deaths in the Castile-Leon region of Spain attributable to cardiovascular disease.

Source: Adapted from Fernandez-Raga et al. (2010).

During the warm half of the year (April–September), they found a rise in temperature had an inverse or protective effect with respect to CVD mortality (a 1% decrease in death in response to a 1°C increase in apparent temperature). This finding is unusual but also has been observed in Dublin, Ireland, as reported by Baccini et al. (2008, 2011). Wichmann et al. found no association with RD and CBD mortality. At the other end of the thermal spectrum, during the cold half of the year, all three associations were inverse or protective. This finding, according to the researchers, is "consistent with other studies (Eurowinter Group, 1997; Nafstad et al., 2001; Braga et al., 2002; O'Neill et al., 2003; Analitis et al., 2008)."
Matzarakis et al. (2011) studied the relationship between heat stress and all-cause mortality in the densely populated city of Vienna (Austria). Based on data from 1970–2007, and after adjusting the long-term mortality rate to account for temporal variations in the size of the population of Vienna, temporal changes in life expectancy, and the changing age structure of Vienna's population, the three researchers found a significant relationship between heat stress and mortality. However, over this 38-year period, "some significant decreases of the sensitivity were found, especially in the medium heat stress levels," they report. These decreases in sensitivity, they write, "could indicate active processes of long-term adaptation to the increasing heat stress." In the discussion section of their paper, they write such sensitivity changes "were also found for other regions," citing Davis et al. (2003), Koppe (2005), Tan et al. (2007), and Donaldson and Keatinge (2008). In the conclusion of their paper, they refer to these changes as

"positive developments."
Kysely and Plavcova then examined "temporal changes in mortality associated with spells of large positive temperature anomalies (hot spells) in extended summer season in the population of the Czech Republic (Central Europe) during 1986–2009." They found declining mortality trends in spite of rising temperature trends, just the opposite of what IPCC claims will occur in response to global warming. The Czech scientists add, "the finding on reduced vulnerability of the population remains unchanged if possible confounding effects of within- season acclimatization and mortality displacement are taken into account," and "neither does it depend on the changing age structure of the population, since similar (and slightly more pronounced) declines in the mortality impacts are found in the elderly (age group 70+ years) when examined separately."

References

Alberdi, J.C., Diaz, J., Montero, J.C., and Miron, I. 1998. Daily mortality in Madrid community 1986–1992: relationship with meteorological variables. European Journal of Epidemiology 14: 571–578.

Analitis, A., Katsouyanni, K., Biggeri, A., Baccini, M., Forsberg, B., Bisanti, L., Kirchmayer, U., Ballester, F., Cadum, E., Goodman, P.B., Hojs, A., Sunyer, J., Tiittanen, P., and Michelozzi, P.
2008. Effects of cold weather on mortality: Results from 15 European cities within the PHEWE project. American Journal of Epidemiology 168: 1397–1408.

Baccini, M., Biggeri, A., Accetta, G., Kosatsky, T., Katsouyanni, K., Analitis, A., Anderson, H.R., Bisanti, L., D'Ippoliti, D., Danova, J., Forsberg, B., Medina, S., Paldy, A., Rabczenko, D., Schindler, C., and Michelozzi, P. 2008. Heat effects on mortality in 15 European cities. Epidemiology 19: 711–719.

Baccini, M., Tom, K., and Biggeri, A. 2011. Impact of heat on mortality in 15 European cities: Attributable deaths under different weather scenarios. Journal of Epidemiology and Community Health 65: 64–70.

Ballester, F., Corella, D., Perez-Hoyos, S., and Saez, M. 1997. Mortality as a function of temperature. A study in Valencia, Spain, 1991–1993. International Journal of Epidemiology 26: 551–561.

Beniston, M. 2002. Climatic change: possible impact on human health. Swiss Medical Weekly 132: 332–337.

Bi, P. and Walker, S. 2001. Mortality trends for deaths related to excessive heat (E900) and excessive cold (E901), Australia, 1910–1997. Environmental Health 1: 80–86.

Braga, A., Zanobetti, A., and Schwartz, J. 2002. The effect of weather on respiratory and cardiovascular deaths in 12 US cities. Environmental Health Perspectives 110: 859–863.

Christidis, N., Donaldson, G.C., and Stott, P.A. 2010. Causes for the recent changes in cold- and heat-related mortality in England and Wales. Climatic Change 102: 539–553.

Curriero, F.C., Heiner, K.S., Samet, J.M., Zeger, S.L., Strug, L., and Patz, J.A. 2002. Temperature and mortality in 11 cities of the Eastern United States. American Journal of Epidemiology 155: 80–87.

Davis, R.E., Knappenberger, P.C., Novicoff, W.M., and Michaels, P.J. 2002. Decadal changes in heat-related human mortality in the Eastern US. Climate Research 22: 175–184.

Davis, R.E., Knappenberger, P.C., Novicoff, W.M,. and Michaels, P.J. 2003a. Decadal changes in summer mortality in U.S. cities. International Journal of Biometeorology 47: 166–175.

Davis, R.E., Knappenberger, P.C., Michaels, P.J., and Novicoff, W.M. 2003b. Changing heat- related mortality in the United States. Environmental Health Perspectives 111: 1712–1718.

Diaz, J., Garcia, R., Lopez, C., Linares, C., Tobias, A., and Prieto, L. 2005. Mortality impact of extreme winter temperatures. International Journal of Biometeorology 49: 179–183.

Donaldson, G.C. and Keatinge, W.R. 2008. Direct effects of rising temperatures on mortality in the UK. In: Kovats, R.S. (Ed.) Health Effects of Climate Change in the UK 2008: An Update of the Department of Health Report 2001/2002. Department of Health, United Kingdom, pp. 81–90.

Donaldson, G.C., Kovats, R.S., Keatinge, W.R., and McMichael, A.J. 2001. Heat- and cold- related mortality and morbidity and climate change. In: Maynard, R.L. (Ed.) Health Effects of Climate Change in the UK. Department of Health, London, UK, pp. 70–80.

Eccles, R. 2002. An explanation for the seasonality of acute upper respiratory tract viral infections. Acta Oto-Laryngologica 122: 183–191.

Eng, H. and Mercer, J.B. 1998. Seasonal variations in mortality caused by cardiovascular diseases in Norway and Ireland. Journal of Cardiovascular Risk 5: 89–95.

Eurowinter Group. 1997. Cold exposure and winter mortality from ischaemic heart disease, cerebrovascular disease, respiratory disease, and all causes in warm and cold regions of Europe. The Lancet 349: 1341–1346.

Fernandez-Raga, M., Tomas, C., and Fraile, R. 2010. Human mortality seasonality in Castile-

Leon, Spain, between 1980 and 1998: the influence of temperature, pressure and humidity. International Journal of Bio-meteorology 54: 379–392.

Fleming, D.M., Cross, K.W., Sunderland, R., and Ross, A.M. 2000. Comparison of the seasonal patterns of asthma identified in general practitioner episodes, hospital admissions, and deaths. Thorax 55: 662–665.

Fouillet, A., Rey, G., Wagner, V., Laaidi, K., Empereur-Bissonnet, P., Le Tertre, A., Frayssinet, P., Bessemoulin, P., Laurent, F., De Crouy-Chanel, P., Jougla, E., and Hemon, D. 2008. Has the impact of heat waves on mortality changed in France since the European heat wave of summer 2003? A study of the 2006 heat wave. International Journal of Epidemiology 37: 309–317.

Garssen, J., Harmsen, C., and de Beer, J. 2005. The effect of the summer 2003 heat wave on mortality in the Netherlands. Euro Surveillance 10: 165–168.

Gouveia, N., Hajat, S., and Armstrong, B. 2003. Socioeconomic differentials in the temperature- mortality relationship in Sao Paulo, Brazil. International Journal of Epidemiology 32: 390–397.

Grech, V., Balzan, M., Asciak, R.P., and Buhagiar, A. 2002. Seasonal variations in hospital admissions for asthma in Malta. Journal of Asthma 39: 263–268.

Keatinge, W.R. and Donaldson, G.C. 2001. Mortality related to cold and air pollution in London after allowance for effects of associated weather patterns. Environmental Research 86: 209–216.

Keatinge, W.R., Donaldson, G.C., Bucher, K., Jendritzky, G., Cordioli, E., Martinelli, M., Katsouyanni, K., Kunst, A.E., McDonald, C., Nayha, S., and Vuori, I. 2000b. Winter mortality in relation to climate. International Journal of Circumpolar Health 59: 154–159.

Koppe, C. 2005. Gesundheitsrelevante Bewertung von thermischer Belastung unter Berucksichtigung der kurzfristigen Anpassung der Bevolkerung an die lokalen Witterungsverhaltnisse. Albert-Ludwigs-University of Freiburg, Germany.

Kunst, A.E., Looman, W.N.C., and Mackenbach, J.P. 1993. Outdoor temperature and mortality in the Netherlands: a time-series analysis. American Journal of Epidemiology 137: 331–341.

Kysely, J. and Huth, R. 2004. Heat-related mortality in the Czech Republic examined through synoptic and 'traditional' approaches. Climate Research 25: 265–274.

Kysely, J. and Plavcova, E. 2012. Declining impacts of hot spells on mortality in the Czech Republic, 1986–2009: adaptation to climate change? Climatic Change 113: 437–453.

Laaidi, M., Laaidi, K., and Besancenot, J.-P. 2006. Temperature-related mortality in France, a comparison between regions with different climates from the perspective of global warming. International Journal of Biometeorology 51: 145–153.

Law, B.J., Carbonell-Estrany, X., and Simoes, E.A.F. 2002. An update on respiratory syncytial

virus epidemiology: a developed country perspective. Respiratory Medicine Supplement B 96: S1–S2.

Martens, P. and Huynen, M. 2001. Will global climate change reduce thermal stress in the Netherlands? Epidemiology 12: 753–754.

Matthies, F. and Menne, B. 2009. Prevention and management of health hazards related to heatwaves. International Journal of Circumpolar Health 68: 8–22.

Matzarakis, A., Muthers, S., and Koch, E. 2011. Human biometeorological evaluation of heat- related mortality in Vienna. Theoretical and Applied Climatology 105: 1–10.

Mayer, H. and Hoppe, P. 1987. Thermal comfort of man in different urban environments. Theoretical and Applied Climatology 38: 43–49.

Muggeo, V.M.R. and Vigotti, M.A. 2002. Modelling trend in break-point estimation: an assessment of the heat tolerance and temperature effects in four Italian cities. In: Stasinopoulos, M. and Touloumi, G. (Eds.) Proceedings of the 17th International Workshop on Statistical Modelling, University of North London, Chania, Greece, pp. 493–500.

Nafstad, P., Skrondal, A., and Bjertness, E. 2001. Mortality and temperature in Oslo, Norway, 1990–1995. European Journal of Epidemiology 17: 621–627.

O'Neill, M.S., Zanobetti, A., and Schwartz, J. 2003. Modifiers of the temperature and mortality association in seven US cities. American Journal of Epidemiology 157: 1074–1082.

Rooney, C., McMichael, A.J., Kovats, R.S., and Coleman, M.P. 1998. Excess mortality in England and Wales, and in Greater London, during the 1995 heat wave. Journal of Epidemiology and Community Health 52: 482–486.

Sheridan, S.C., Kalkstein, A.J., and Kalkstein, L.S. 2009. Trends in heat-related mortality in the United States, 1975–2004. Natural Hazards 50: 145–160.

Tan, J., Zheng, Y., Tang, X., Guo, C., Li, L., Song, G., Zhen, X., Yuan, D., Kalkstein, A., and Chen, H. 2007. Heat wave impacts on mortality in Shanghai 1998 and 2003. International Journal of Biometeorology 51: 193–200.

Verlato, G., Calabrese, R., and De Marco, R. 2002. Correlation between asthma and climate in the European Community Respiratory Health Survey. Archives of Environmental Health 57: 48– 52.

Wichmann, J., Anderson, Z.J., Ketzel, M., Ellermann, T., and Loft, S. 2011. Apparent temperature and cause-specific mortality in Copenhagen, Denmark: A case-crossover analysis. International Journal of Environmental Research and Public Health 8: 3712–3727.

Observational Research in North America

Goklany and Straja (2000) examined trends in United States death rates over the period 1979– 1997 due to excessive hot and cold weather. They report there were no trends in deaths due to either extreme heat or cold in the entire population or in the older, more-susceptible age groups, those aged 65 and over, 75 and over, and 85 and over. Deaths due to extreme cold in these older age groups exceeded those due to extreme heat by as much as 80% to 125%. With respect to the absence of trends in death rates attributable to either extreme heat or cold, Goklany and Straja say this "suggests that adaptation and technological change may be just as important determinants of such trends as more obvious meteorological and demographic factors."
Davis et al. (2003) evaluated "annual excess mortality on days when apparent temperatures—an index that combines air temperature and humidity—exceeded a threshold value for 28 major metropolitan areas in the United States from 1964 through 1998." They found "for the 28-city average, there were 41.0 ± 4.8 excess heat-related deaths per year (per standard million) in the 1960s and 1970s, 17.3 ± 2.7 in the 1980s, and 10.5 ± 2.0 in the 1990s," a remarkable decline. They conclude, "heat-related mortality in the United States seems to be largely preventable at present."
Davis et al. (2004) examined the seasonality of mortality due to all causes, using monthly data for 28 major U.S. cities from 1964 to 1998, and then calculated the consequences of a future 1°C warming of the conglomerate of those cities. At all locations studied, they report "warmer months have significantly lower mortality rates than colder months." They calculate "a uniform 1°C warming results in a net mortality decline of 2.65 deaths (per standard million) per metropolitan statistical area" (emphasis added). The primary implication of Davis et al.'s findings, in their words, "is that the seasonal mortality pattern in US cities is largely independent of the climate and thus insensitive to climate fluctuations, including changes related to increasing greenhouse gases."
Deschenes and Moretti (2009) analyzed the relationship between weather and mortality, based on "data that include the universe of deaths in the United States over the period 1972– 1988," in which they "match each death to weather conditions on the day of death and in the county of occurrence." They discovered "hot temperature shocks are indeed associated with a large and immediate spike in mortality in the days of the heat wave," but "almost all of this excess mortality is explained by near-term displacement." As a result, "in the weeks that follow a heat wave, we find a marked decline in mortality hazard, which completely offsets the increase during the days of the heat wave," so "there is virtually no lasting impact of heat waves on mortality." In the case of cold temperature days, they also found "an immediate spike in mortality but "there is no offsetting decline in the weeks that follow," so "the cumulative effect of one day of extreme cold temperature during a thirty-day window is an increase in daily mortality by as much as 10%."

References

Davis, R.E., Knappenberger, P.C., Michaels, P.J., and Novicoff, W.M. 2003. Changing heat- related mortality in the United States. Environmental Health Perspectives 111: 1712–1718.

Davis, R.E., Knappenberger, P.C., Michaels, P.J., and Novicoff, W.M. 2004. Seasonality of climate-human mortality relationships in US cities and impacts of climate change. Climate Research 26: 61–76.

Davis, R.E., Knappenberger, P.C., Novicoff, W.M., and Michaels, P.J. 2002. Decadal changes in heat-related human mortality in the eastern United States. Climate Research 22: 175–184.

Deschenes, O. and Moretti, E. 2009. Extreme weather events, mortality, and migration. TheReview of Economics and Statistics 91: 659–681.

Fischer, P.H., Brunekreef, B., and Lebret, E. 2004. Air pollution related deaths during the 2003 heat wave in the Netherlands. Atmospheric Environment 38: 1083–1085.

Goklany, I.M. and Straja, S.R. 2000. U.S. trends in crude death rates due to extreme heat and cold ascribed to weather, 1979–97. Technology 7S: 165–173.

O'Neill, M.S., Hajat, S., Zanobetti, A., Ramierz-Aguilar, M., and Schwartz, J. 2005. Impact of control for air pollution and respiratory epidemics on the estimated associations of temperature and daily mortality. International Journal of Biometeorology 50: 121–129.

Stedman, J.R. 2004. The predicted number of air pollution related deaths in the UK during the August 2003 heatwave. Atmospheric Environment 38: 1087–1090.

Global Warming and Cardiovascular Disease

The key findings are that

- Global warming, if it does occur, would reduce the incidence of fatal coronary events related to low temperatures and wintry weather by a much greater degree than it increases the incidence of death or serious heat related events associated with high temperatures and summer heat waves.

- Non-fatal myocardial infarction is also less frequent during unseasonably warm periods than during unseasonably cold periods.

- Any cost-benefit analysis that attributes an increase in cardiovascular events to warming is incorrect. Heat illness injures and kills by other means and has a much lesser death toll proportionately than cold related events. Heat illness injury and death in heat waves affects the debilitated and chronically ill in hot unventilated environments and the mechanism is dehydration and loss of core body temperature control.

Cardiovascular diseases affect the heart and or the blood vessels. They include arrhythmia, arteriosclerosis, congenital heart disease, and coronary artery disease, diseases of the aorta and its branches, disorders of the peripheral vascular system, endocarditis, heart valve disease, hypertension, orthostatic hypotension, and

shock. According to IPCC, exposure to rising temperatures and especially heat waves can cause premature deaths due to heat-induced illness. The claims that it causes stroke or myocardial infarctions are not correct except to concede that ultimately most deaths are cardiovascular in nature.
Empirical research suggests that heat illness can cause collapse and death, but the mechanism is fluid and circulatory collapse, not stroke or heart attack. Heat stroke is severe heat illness with loss of temperature control that produces brain dysfunction; it's not a cerebral thrombosis or hemorrhage, a true stroke.
That aside, the IPCC overlooks the fact that cooler temperatures cause an even larger number of premature deaths, with the result that a warmer world would experience fewer deaths in total due to cardiovascular disease.

Global Warming and Respiratory Disease

The key findings of this section include the following:

- Global warming, if it did occur would reduce incidence of death due to respiratory disease around the world, for example the Americas, Spain, Canada, Shanghai, and even on the subtropical island of Taiwan.

- Lower minimum temperatures are a strong risk factor for outpatient visits for respiratory diseases. Warmer temperatures reduce rates of respiratory disease.

- Any cost-benefit analysis that attributes increases in deaths or disease and disability or loss of work/school time to warming is incorrect and not a reliable guide for public policy.

Respiratory diseases are diseases affecting the organs and tissues that make gas exchange possible in humans and other higher organisms. They range from the common cold, allergies, asthma, and bronchiolitis to life-threatening conditions including pneumonia, pulmonary embolism, and lung cancer. Acute respiratory disease is a condition in which breathing becomes difficult and oxygen levels in the blood drop lower than normal. Respiratory diseases are widespread. For example, childhood asthma affects more than 300 million people worldwide (Baena-Cagnani and Badellino, 2011). Non-fatal respiratory diseases impose enormous social costs due to days lost from work and school (Mourtzoukou and Falagas, 2007).
According to IPCC, rising atmospheric carbon dioxide concentrations due to the combustion of fossil fuels causes global warming, and this temperature increase causes increased deaths due to respiratory disease. However, examination of real-world data reveals unassailable evidence that colder temperatures cause more deaths and hospital admissions due to respiratory disease than do warmer temperatures.
Some of the studies cited earlier in this chapter on lower death rates due to warmer temperatures and cardiovascular disease also identified specific reductions in fatalities due to respiratory diseases, so their research also appears in this section. Keatinge and Donaldson (2001), for example, studied of the effects of temperature on mortality in people over 50 years of age in the greater London area over the period 1976–1995. Simple plots of mortality rate versus daily air temperature revealed a linear increase in mortality as the air temperature fell from 15°C to near 0°C. Mortality rates at temperatures above 15°C, on the other hand, showed no trend. The authors say it is because "cold causes mortality mainly from arterial thrombosis and respiratory disease, attributable in turn to cold-induced hemo-concentration and hypertension and respiratory infections" (emphasis added).
Nafstad et al. (2001) studied the association between temperature and daily mortality in citizens of Oslo, Norway over the period 1990 to 1995. The results showed the mean daily number of respiratory-related

deaths was considerably higher in winter (October–March) than in summer (April–September). Winter deaths associated with respiratory diseases were 47% more numerous than summer deaths. They conclude, "A milder climate would lead to a substantial reduction in average daily number of deaths." Read milder as warmer.

Hajat and Haines (2002) examined the relationship between cold temperatures and the number of visits by the elderly to general practitioners for asthma, lower respiratory diseases other than asthma, and upper respiratory diseases other than allergic rhinitis as obtained for registered patients aged 65 and older from several London practices between January 1992 and September 1995. They found the mean number of consultations was higher in cool-season months (October–March) than in warm-season months (April–September) for all respiratory diseases. At mean temperatures below 5°C, the relationship between respiratory disease consultations and temperature was linear, and stronger at a time lag of six to 15 days A 1°C decrease in mean temperature below 5°C was associated with a 10.5% increase in all respiratory disease consultations.

Braga et al. (2002) conducted a time-series analysis of both the acute and lagged influence of temperature and humidity on mortality rates in 12 U.S. cities, finding no clear evidence for a link between humidity and respiratory-related deaths. With respect to temperature, they found respiratory-related mortality increased in cities with more variable temperature. This phenomenon, they write, "suggests that increased temperature variability is the most relevant change in climate for the direct effects of weather on respiratory mortality."

Gouveia et al. (2003) extracted daily counts of deaths from all causes, except violent

deaths and neonatal deaths (up to one month of age), from Sao Paulo, Brazil's mortality information system for the period 1991–1994 and analyzed them for effects of temperature. For respiratory-induced deaths, death rates due to a 1°C cooling were twice as great as death rates due to a 1°C warming in adults and 2.8 times greater in the elderly.

Nakaji et al. (2004) evaluated seasonal trends in deaths due to various diseases in Japan, using nationwide vital statistics from 1970 to 1999 and concurrent mean monthly air temperature data. They found the numbers of deaths due to respiratory diseases, including pneumonia and influenza, rise to a maximum during the coldest time of the year. The team of nine scientists concludes, "To reduce the overall mortality rate and to prolong life expectancy in Japan, measures must be taken to reduce those mortality rates associated with seasonal differences."

Bartzokas et al. (2004) "examined the relationship between hospital admissions for cardio-vascular (cardiac in general including heart attacks) and/or respiratory diseases (asthma etc.) in a major hospital in Athens [Greece] and meteorological parameters for an 8-year period." Over the whole year, they found, "there was a dependence of admissions on temperature," and low temperatures were "responsible for a higher number of admissions." Specifically, "there was a decrease of cardiovascular or/and respiratory events from low to high values [of temperature], except for the highest temperature class in which a slight increase was recorded."

Kovats et al. (2004) studied patterns of temperature-related hospital admissions and deaths in Greater London during the mid-1990s. For the three-year period 1994–1996, they found respiratory-related deaths were nearly 150% greater in the depth of winter cold than at the height of summer warmth. They also found the mortality impact of the heat wave of 29 July to 3 August 1995 (which boosted daily mortality by just over 10%) was so tiny it could not be discerned among the random scatter of plots of three-year-average daily deaths from cardiovascular and respiratory problems versus day of year. Similarly, in a study of temperature effects on mortality in three English counties (Hampshire, West Midlands, and West Yorkshire), McGregor (2005) found "the occurrence of influenza ... helps elevate winter mortality above that of summer."

Carder et al. (2005) investigated the relationship between outside air temperature and deaths due to all non-accident causes in the three largest cities of Scotland (Glasgow, Edinburgh, and Aberdeen) between January 1981 and December 2001. The authors observed "an overall increase in mortality as temperature decreases," which "appears to be steeper at lower temperatures than at warmer temperatures," and "there is little evidence of an increase in mortality at the hot end of the temperature range." Specifically regarding respiratory disease, they found "for temperatures below 11°C, a 1°C drop in the daytime mean temperature on any one day was associated with an increase in respiratory mortality of 4.8% over the following month."
Donaldson (2006) studied the effect of annual mean daily air temperature on the length of the yearly respiratory syncytial virus (RSV) season, the virus which causes bronchiolitis, in England and Wales for 1981–2004. Reporting "climate change may be shortening the RSV season," Donaldson found "the seasons associated with laboratory isolation of respiratory syncytial virus (for 1981–2004) and RSV-related emergency department admissions (for 1990– 2004) ended 3.1 and 2.5 weeks earlier, respectively, per 1°C increase in annual central England temperature ($P = 0.002$ and 0.043, respectively)." Consequently, since "no relationship was observed between the start of each season and temperature," he reports, so "the RSV season has become shorter." He concludes, "These findings imply a health benefit of global warming in England and Wales associated with a reduction in the duration of the RSV season and its consequent impact on the health service."
Frei and Gassner (2008) studied hay fever prevalence in Switzerland from 1926 to 1991, finding it rose from just under 1% of the country's population to just over 14%, but from 1991 to 2000 it leveled off, fluctuating about a mean value on the order of 15%. The authors write, "several studies show that no further increase in asthma, hay fever and atopic sensitization in adolescents and adults has been observed during the 1990s and the beginning of the new century," citing Braun-Fahrlander et al. (2004) and Grize et al. (2006). They write, "Parallel to the increasing hay fever rate, the pollen amounts of birch and grass were increasing from 1969 to 1990," but "subsequently, the pollen of these plant species decreased from 1991 to 2007." They say this finding "is more or less consistent with the changes of the hay fever rate that no longer increased during this period and even showed a tendency to decrease slightly." Nearly identical findings were presented a year later (Frei, 2009). Although some have claimed rising temperatures and CO2 concentrations will lead to more pollen and more hay fever (Wayne et al., 2002), the analyses of Frei (2009) and Frei and Gassner (2008) suggest that is not true of Switzerland.
Miller et al. (2012) extracted annual prevalence data for frequent otitis media (defined as three or more ear infections per year), respiratory allergy, and non-respiratory seizures in children from the U.S. National Health Interview Survey for 1998 to 2006. They also obtained average annual temperatures for the same period from the U.S. Environmental Protection Agency. They found "annual temperature did not influence the prevalence of frequent otitis media," "annual temperature did not influence prevalence of respiratory allergy," and "annual temperature and sex did not influence seizure prevalence." They conclude their findings "may demonstrate that average temperature is not likely to be the dominant cause of the increase in allergy burden or that larger changes in temperatures over a longer period are needed to observe this association." They further conclude, "In the absence of more dramatic annual temperature changes, we do not expect prevalence of otitis media to change significantly as global warming may continue to affect our environment."
Xu et al. (2013) examined the relationship between diurnal temperature range (DTR) and emergency department admissions for childhood asthma in Brisbane, Australia, from January 1st 2003 to December 31st 2009. The six scientists report "childhood asthma increased above a DTR of 10°C" and "was the greatest for lag 0–9 days, with a 31% increase in [hospital] emergency department admissions per 5°C increment of DTR," further noting, "male children and children aged 5–9 years appeared to be more vulnerable to the DTR effect than others."

Ge et al. (2013) also investigated respiratory health and DTR. The researchers collected numbers of daily emergency-room visits for RTI at one of the largest medical establishments in Shanghai, China (Huashan Hospital) between 1 January 2008 and 30 June 2009, along with DTR data and data pertaining to possible confounding air pollutants (PM10, SO2, and NO2). After making appropriate statistical analyses, the scientists determined increasing DTRs were closely associated with daily emergency-room visits for RTIs, such that "an increase of 1°C in the current-day and in the 2-day moving average DTR corresponded to a 0.94% and 2.08% increase in emergency-room visits for RTI, respectively."
Lin et al. (2013) used data on daily area-specific deaths from all causes, circulatory diseases, and respiratory diseases in Taiwan, developing relationships between each of these cause-of-death categories and a number of cold-temperature related parameters for 2000–2008. The five researchers discovered "mortality from [1] all causes and [2] circulatory diseases and [3] outpatient visits of respiratory diseases has a strong association with cold temperatures in the subtropical island, Taiwan." In addition, they found "minimum temperature estimated the strongest risk associated with outpatient visits of respiratory diseases."

References

Baena-Cagnani, C. and Badellino, H. 2011. Diagnosis of allergy and asthma in childhood. Current Allergy and Asthma Reports 11: 71–77.

Bartzokas, A., Kassomenos, P., Petrakis, M., and Celessides, C. 2004. The effect of meteorological and pollution parameters on the frequency of hospital admissions for cardiovascular and respiratory problems in Athens. Indoor and Build Environment 13: 271–275.

Braga, A.L.F., Zanobetti, A., and Schwartz, J. 2002. The effect of weather on respiratory and cardiovascular deaths in 12 U.S. cities. Environmental Health Perspectives 110: 859–863.

Braun-Fahrlander, C., Gassner, M., Grize, L., Takken-Sahli, K., Neu, U., Stricker, T., Varonier, H.S., Wuthrich, B., Sennhauser, F.H., and SCARPOL Team. 2004. No further increase in asthma, hay fever and atopic sensitization in adolescents living in Switzerland. European Respiratory Journal 23: 407–413.

Carder, M., McNamee, R., Beverland, I., Elton, R., Cohen, G.R., Boyd, J., and Agius, R.M. 2005. The lagged effect of cold temperature and wind chill on cardiorespiratory mortality in Scotland. Occupational and Environmental Medicine 62: 702–710.

Donaldson, G.C. 2006. Climate change and the end of the respiratory syncytial virus season. Clinical Infectious Diseases 42: 677–679.

Easterling, D.R., Horton, B., Jones, P.D., Peterson, T.C., Karl, T.R., Parker, D.E., Salinger, M.J., Razuvayev, V., Plummer, N., Jamason, P., and Folland, C.K. 1997. Maximum and minimum temperature trends for the globe. Science 277: 364–367.

Frei, T. 2009. Trendwende bei der Pollinose und dem Pollenflug? Allergologie 32: 123–127.

Frei, T. and Gassner, E. 2008. Trends in prevalence of allergic rhinitis and correlation with pollen counts in Switzerland. International Journal of Biometeorology 52: 841–847.

Ge, W.Z., Xu, F., Zhao, Z.H., Zhao, J.Z., and Kan, H.D. 2013. Association between diurnal temperature range and respiratory tract infections. Biomedical and Environmental Sciences 26: 222–225.

Gouveia, N., Hajat, S., and Armstrong, B. 2003. Socioeconomic differentials in the temperature- mortality relationship in Sao Paulo, Brazil. International Journal of Epidemiology 32: 390–397.

Grize, L., Gassner, M., Wuthrich, B., Bringolf-Isler, B., Takken-Sahli, K., Sennhauser, F.H., Stricker, T., Eigenmann, P.A., Braun-Fahrlander, C., and SCARPOL Team. 2006. Trends in prevalence of asthma, allergic rhinitis and atopic dermatitis in 5–7-year old Swiss children from 1992 to 2001. Allergy 61: 556–562.

Hajat, S. and Haines, A. 2002. Associations of cold temperatures with GP consultations for respiratory and cardiovascular disease amongst the elderly in London. International Journal of Epidemiology 31: 825–830.

Jato, V., Rodriguez-Rajo, F.J., Seijo, M.C., and Aira, M.J. 2009. Poaceae pollen in Galicia (N.W. Spain): characterization and recent trends in atmospheric pollen season. International Journal of Biometeorology 53: 333–344.

Keatinge, W.R. and Donaldson, G.C. 2001. Mortality related to cold and air pollution in London after allowance for effects of associated weather patterns. Environmental Research 86: 209–216.

Kovats, R.S., Hajat, S., and Wilkinson, P. 2004. Contrasting patterns of mortality and hospital admissions during hot weather and heat waves in Greater London, UK. Occupational and Environmental Medicine 61: 893–898.

Lin, Y.-K., Wang, Y.-C., Lin, P.-L., Li, M.-H., and Ho, T.-J. 2013. Relationships between cold- temperature indices and all causes and cardiopulmonary morbidity and mortality in a subtropical island. Science of the Total Environment 461–462: 627–635.

McGregor, G.R. 2005. Winter North Atlantic Oscillation, temperature and ischaemic heart disease mortality in three English counties. International Journal of Biometeorology 49: 197– 204.

Miller, M.E., Shapiro, N.L. and Bhattacharyya, N. 2012. Annual temperature and the prevalence of frequent ear infections in childhood. American Journal of Otolaryngology - Head and Neck Medicine and Surgery 33: 51-55.

Mourtzoukou, E.G. and Falagas, M.E. 2007. Exposure to cold and respiratory tract infections. International Journal of Tuberculosis and Lung Disease 11: 938–943.

Nafstad, P., Skrondal, A., and Bjertness, E. 2001. Mortality and temperature in Oslo, Norway.

1990–1995. European Journal of Epidemiology 17: 621–627.

Nakaji, S., Parodi, S., Fontana, V., Umeda, T., Suzuki, K., Sakamoto, J., Fukuda, S., Wada, S., and Sugawara, K. 2004. Seasonal changes in mortality rates from main causes of death in Japan (1970–1999). European Journal of Epidemiology 19: 905–913.

Wang, Y.C., Lin, Y.K., Chuang, C.Y., Li, M.H., Chou, C.H., Liao, C.H., and Sung, F.C. 2012. Associating emergency room visits with first and prolonged extreme temperature event in Taiwan: a population-based cohort study. Science of the Total Environment 416: 97–104.

Wayne, P., Foster, S., Connolly, J., Bazzaz, F., and Epstein, P. 2002. Production of allergenic pollen by ragweed (Ambrosia artemisiifolia L.) is increased in CO2-enriched atmospheres.
Annals of Allergy, Asthma, and Immunology 88: 279–282.

Xu, Z., Huang, C., Su, H., Turner, L.R., Qiao, Z., and Tong, S. 2013. Diurnal temperature range and childhood asthma: a time-series study. Environmental Health 12: 10.1186/1476-069X-12-12.

Global Warming and Strokes

The key findings of this section include the following:

- Any warming would reduce the incidence of death due to stroke in many parts of the world, including Russia, Korea, Japan, Africa, Asia, Europe, Latin America, and the Caribbean.

- Low minimum temperatures are a stronger risk factor than high temperatures for stroke incidence and hospitalization.

- Any cost-benefit analysis that attributes increased strokes to a prediction of global warming is incorrect and not a reliable guide for public policy.

A stroke occurs when blood flow to an area in the brain is cut off. Ischemic stroke occurs when clots form in the brain's blood vessels, in blood vessels leading to the brain, or in blood vessels elsewhere in the body and then travel to the brain. Ischemic stroke can also occur when too much plaque (fatty deposits and cholesterol) clogs the brain's blood vessels. Hemorrhagic strokes occur when a blood vessel in the brain breaks or ruptures. The result is blood seeping into the brain tissue, causing damage to brain cells. The most common causes of hemorrhagic stroke are high blood pressure and brain aneurysms. An aneurysm is a bulge in a blood vessel caused by a weakness and thinning of the blood vessel wall. Aneurysms are prone to burst and a major cause of hemorrhagic stroke (WebMD, 2015).
According to IPCC, rising atmospheric carbon dioxide concentrations due to the combustion of fossil fuels causes global warming, and this temperature increase causes increased deaths due to strokes. Not true. Examination of real-world data reveals unseasonable cold temperatures cause more deaths and hospital admissions due to stroke than do unseasonable warm temperatures.
Feigin et al. (2000) examined the relationship between the incidence of stroke and ambient temperatures over the period 1982-1993 in Novosibirsk, Siberia, which has one of the highest stroke incidence rates in the world. Based on analyses of 2,208 patients with sex and age distributions similar to those of Russia as a whole, they found a statistically significant association between stroke and low ambient temperature. In the

case of ischemic stroke (IS), which accounted for 87% of all stroke types, they determined "the risk of IS occurrence on days with low ambient temperature [was] 32% higher than that on days with high ambient temperature." They conclude the "very high stroke incidence in Novosibirsk, Russia may partially be explained by the highly prevalent cold factor there." There is no reason to believe that temperature variations would have a discernible effect on hemorrhagic strokes that occur because of vascular pathology, not occlusion.

Hong et al. (2003) investigated the association between the onset of ischemic stroke and prior episodic decreases in temperature in 545 patients who suffered strokes in Incheon, Korea from January 1998 to December 2000. They report "decreased ambient temperature was associated with risk of acute ischemic stroke," with the strongest effect being seen on the day after exposure to cold weather, further noting "even a moderate decrease in temperature can increase the risk of ischemic stroke." They also found "risk estimates associated with decreased temperature were greater in winter than in the summer," which suggests "low temperatures as well as temperature changes are associated with the onset of ischemic stroke." Finally, they explain the reason for the 24- to 48-hour lag between exposure to cold and the onset of stroke "might be that it takes some time for the decreasing temperature to affect blood viscosity or coagulation.

Nakaji et al. (2004) evaluated seasonal trends in deaths due to various diseases in Japan using nationwide vital statistics from 1970 to 1999 together with mean monthly temperature data. They found the peak mortality rate due to stroke was two times greater in winter (January) than at the time of its yearly minimum (August and September).

Chang et al. (2004) analyzed data from the World Health Organization (WHO) Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception (WHO, 1995) to determine the effects of monthly mean temperature on rates of hospitalization for arterial stroke and acute myocardial infarction among women aged 15–49 from 17 countries in Africa, Asia, Europe, Latin America, and the Caribbean. They found among these women, a 5°C reduction in mean air temperature was associated with a 7% increase in the expected hospitalization rate due to stroke, and this effect was relatively acute, within a period of about a month, the scientists write.

Gill et al. (2012) write, "in the past two decades, several studies reported that meteorologic changes are associated with monthly and seasonal spikes in the incidence of aneurysmal subarachnoid hemorrhage (aSAH)," and "analysis of data from large regional databases in both hemispheres has revealed increased seasonal risk for aSAH in the fall, winter and spring," citing among other sources Feigin et al. (2001), Abe et al. (2008), and Beseoglu et al. (2008). Gill et al. identified the medical records of 1,175 patients at the Johns Hopkins Hospital in Baltimore, Maryland (USA) who were admitted with a radiologically confirmed diagnosis of aSAH between 1 January 1991 and 1 March 2009. The six scientists report both "a one-day decrease in temperature and colder daily temperatures were associated with an increased risk of incident aSAH," and "these variables appeared to act synergistically" and were "particularly predominant in the fall, when the transition from warmer to colder temperatures occurred." Gill et al. add their study "is the first to report a direct relationship between a temperature decrease and an increased risk of aSAH," and "it also confirms the observations of several reports of an increased risk of aSAH in cold weather or winter," citing Nyquist et al. (2001) and other sources. Authors' note: This study and others the authors of the study reference are outliers in the sense that they tally aneurysmal sub arachnoid hemorrhage, a different kind of stroke than ischemic strokes, so there is no "mechanism" of coagulation and clot formation that would relate to temperature that might be hypothesized as a cause of cold or cool to cause hemorrhagic stroke.

The reader should be informed that hemorrhagic stroke is because of a different mechanism, the rupture of a weakened wall of a blood vessel, often associated with a bulge called an aneurysm, as opposed to ischemic stroke discussed above that occur because of a blood clot in the brain blood vessel. However the temperature

effect is the same, cold produces an increase in hemorrhagic strokes in addition to its effect on the rate of ischemic strokes.

References

Abe, T., Ohde, S., Ishimatsu, S., Ogata, H., Hasegawa, T., Nakamura, T., and Tokuda, Y. 2008. Effects of meteorological factors on the onset of subarachnoid hemorrhage: a time-series analysis. Journal of Clinical Neuroscience 15: 1005–1010.

Beseoglu, K., Hanggi, D., Stummer, W., and Steiger, H.J. 2008. Dependence of subarachnoid hemorrhage on climate conditions: a systematic meteorological analysis from the Dusseldorf metropolitan area. Neurosurgery 62: 1033–1038.

Chang, C.L., Shipley, M., Marmot, M., and Poulter, N. 2004. Lower ambient temperature was associated with an increased risk of hospitalization for stroke and acute myocardial infarction in young women. Journal of Clinical Epidemiology 57: 749–757.

Feigin, V.L., Anderson, C.S., Anderson, N.E., Broad, J.B., Pledger, M.J., and Bonita, R. 2001. Is there a temporal pattern to the occurrence of subarachnoid hemorrhage in the southern hemisphere? Pooled data from 3 large, population-based incidence studies in Australasia, 1981 to 1997. Stroke 32: 613–619.

Feigin, V.L., Nikitin, Yu.P., Bots, M.L., Vinogradova, T.E., and Grobbee, D.E. 2000. A population-based study of the associations of stroke occurrence with weather para-meters in Siberia, Russia (1982–92). European Journal of Neurology 7: 171–178.

Gill, R.S., Hambridge, H.L., Schneider, E.B., Hanff, T., Tamargo, R.J., and Nyquist, P. 2012. Falling temperature and colder weather are associated with an increased risk of Aneurysmal Subarachnoid Hemorrhage. World Neuro-surgery 79: 136–142.

Hong, Y-C., Rha, J-H., Lee, J-T., Ha, E-H., Kwon, H-J., and Kim, H. 2003. Ischemic stroke associated with decrease in temperature. Epidemiology 14: 473–478.

Nakaji, S., Parodi, S., Fontana, V., Umeda, T., Suzuki, K., Sakamoto, J., Fukuda, S., Wada, S.,

and Sugawara, K. 2004. Seasonal changes in mortality rates from main causes of death in Japan (1970–1999). European Journal of Epidemiology 19: 905–913.

Nyquist, P.A., Brown Jr., R.D., Wiebers, D.O., Crowson, C.S., and O'Fallon, W.M. 2001. Circadian and seasonal occurrence of subarachnoid and intracerebral hemorrhage. Neurology 56: 190–193.

WebMD, 2015. Heart disease and stroke. Website last visited September 22. http://www.webmd.com/heart-disease/stroke.

WHO. 1995. WHO Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception. A multi-national case-control study of cardiovascular disease and steroid hormone contraceptives: description and validation of methods. Journal of Clinical Epidemiology 48: 1513–1547.

Global Warming and Insect-borne Diseases

The key findings of this section include the following:

- Research contradicts the claim that malaria will expand across the globe and intensify as a result of any possible warming.

- Concerns over large increases in dengue fever as a result of rising temperatures are unfounded and unsupported by the scientific literature, as climatic indices are poor predictors for dengue fever infection rates.

- Climate change has not been a significant factor driving the recent temporal patterns in the epidemiology of tick-borne diseases. Ticks are endemic at many latitudes.

The latest IPCC report, the Fifth Assessment Report (AR5) backs down from previous predictions that global warming would facilitate the spread of insect-borne diseases including malaria, dengue fever, and tick-borne diseases. The full report from Working Group II on the subject (IPCC, 2014a, Chapter 11, pp. 722-726) repeatedly admits there is no evidence that climate change has affected the range of vector-borne diseases including tick-borne diseases. However, the Summary for Policymakers inexplicably warns "Throughout the 21st century, climate change is expected to lead to increases in ill-health in many regions and especially in developing countries with low income, as compared to a baseline without climate change (high confidence)." Among the "examples" given is "vector-borne diseases (medium confidence)" (IPCC, 2014b, pp. 19-20). Such predictions are not supported by the evidence.

In a research report in Science, Rogers and Randolph (2000) note "predictions of global climate change have stimulated forecasts that vector-borne diseases will spread into regions that are at present too cool for their persistence." However, the effect of warmer temperatures on insect-borne diseases is complex, sometimes working in favor of and sometimes against the spread of a disease. For example, ambient temperature has historically not determined the range of insect-borne diseases, hotter weather shortens the lifespan of mosquitos, and human adaptation as well as vector control measures can neutralize any detrimental effect of warming, to overwhelm the role of climate. Even those who support IPCC, such as Marm Kilpatrick, an assistant professor in ecology and evolutionary biology at the University of California, Santa Cruz, admits "It's a little bit tricky to make a solid prediction" (Irfan, 2011).

Gething et al. (2010), writing specifically about malaria, may have put it best when they said there has been "a decoupling of the geographical climate-malaria relationship over the twentieth century, indicating that non-climatic factors have profoundly confounded this relationship over time." They note "non-climatic factors, primarily direct disease control and the indirect effects of a century of urbanization and economic development, although spatially and temporally variable, have exerted a substantially greater influence on the geographic extent and intensity of malaria worldwide during the twentieth century than have climatic factors." As for the future, they conclude climate-induced effects "can be offset by moderate increases in coverage levels of currently available interventions."

This section investigates the reliability of IPCC's claim with respect to the three main kinds of insect-borne diseases: malaria, dengue fever, and tick-borne diseases According to the results of a vast body of scientific examination and research on this topic, there is little support for the claims appearing in the latest IPCC Summary for Policymakers.

References

Gething, P.W., Smith, D.L., Patil, A.P., Tatem, A.J., Snow, R.W., and Hay, S.I. 2010. Climate change and the global malaria recession. Nature 465: 342–345.
IPCC. 2014a. Climage Change 2014: Impacts, Adaptation, and Vulnerabilities, Contribution of Working Group II. Chapter 11: Human health: Impacts, adaptations, and co-benefits. New York, NY: Cambridge University Press.

IPCC. 2014b. Summary for Policymakers. In Climage Change 2014: Impacts, Adaptation, and Vulnerabilities, Contribution of Working Group II. New York, NY: Cambridge University Press.

Irfan, U. 2011. Climate change may make insect-borne diseases harder to control. Scientific American. Website, November 21, http://www.scientificamerican.com/article/climate-change- may-make-insect-born-diseases-harder-control/. Last viewed on October 30, 2015.

Rogers, D.J. and Randolph, S.E. 2000. The global spread of malaria in a future, warmer world. Science 289: 1763–1766.

Malaria

A vast body of scientific examination and research contradict the claim that malaria will expand across the globe and intensify as a result of CO2-induced warming.

Jackson et al. (2010) say "malaria is one of the most devastating vector-borne parasitic diseases in the tropical and subtropical regions of the world," noting it affects more than 100 countries.

According to the World Health Organization, Africa carries the highest infection burden of any continent, with nearly 200 million cases reported in 2006, and the Centers for Disease Control and Prevention estimates between 700,000 and 2.7 million people each year die from the dreaded disease (Suh et al., 2004). In addition, Jackson et al. report "the African region bears 90% of these estimated worldwide deaths," and "three-quarters of all malaria related deaths are among African children," citing Breman (2001). According to Reiter (2000), claims that malaria resurgence is the product of CO2-induced global warming ignore other important factors and disregard known facts. A historical analysis of malaria trends, for example, reveals this disease was an important cause of illness and death in England during a period of colder-than-present temperatures throughout the Little Ice Age. Its transmission began to decline only in the nineteenth century, during a warming phase, when, according to Reiter, "temperatures were already much higher than in the Little Ice Age." In short, malaria was prevalent in Europe during some of the coldest centuries of the past millennium, and it has only recently undergone widespread decline, when temperatures have been warming.

Clearly, there are other factors at work in regards to malaria that are more important than temperature. Such factors include the quality of public health services, irrigation and agricultural activities, land use practices, civil strife, natural disasters, ecological change, population change, use of insecticides, and the movement of people (Reiter, 2000; Reiter, 2001; Hay et al., 2002).

Nevertheless, concerns have lingered about the possibility of widespread future increases in malaria due to global warming. These concerns are generally rooted in climate models that typically use only one, or at most two, climate variables in making their predictions of the future distribution of the disease over Earth, and they generally do not include any of the non-climatic factors listed in the preceding paragraph. When more variables are included, a less-worrisome future is projected.

In one modeling study, for example, Rogers and Randolph (2000) employed five climate variables and obtained very different results. Briefly, they used the present-day distribution of malaria to determine the specific climatic constraints that best define that distribution, after which the multivariate relationship they derived from this exercise was applied to future climate scenarios derived from state-of-the-art climate models, in order to map potential future geographical distributions of the disease.

Their study revealed very little change: a 0.84% increase in potential malaria exposure under the "medium-high" scenario of global warming and a 0.92% decrease under the "high" scenario. Rogers and Randolph explicitly state their quantitative model "contradicts prevailing forecasts of global malaria expansion" and "highlights the use of multivariate rather than univariate constraints in such applications. They found "climate warming, expressed as a systematic temperature increase over the 85-year period, does not appear to be responsible for an increase in malaria suitability over any region in Africa." They conclude "research on the links between climate change and the recent resurgence of malaria across Africa would be best served through refinements in maps and models of precipitation patterns and through closer examination of the role of nonclimatic influences."

Kuhn et al. (2003) analyzed the determinants of temporal trends in malaria deaths within England and Wales in 1840–1910 and found "a 1°C increase or decrease was responsible for an increase in malaria deaths of 8.3% or a decrease of 6.5%, respectively," which explains "the malaria epidemics in the 'unusually hot summers' of 1848 and 1859." Nevertheless, the long- term near-linear temporal decline in malaria deaths over the period of study, the researchers write, "was probably driven by nonclimatic factors," among which they identify increasing livestock populations (which tend to divert mosquito biting from humans), decreasing acreages of marsh wetlands (where mosquitoes breed), as well as "improved housing, better access to health care and medication, and improved nutrition, sanitation, and hygiene." Kuhn et al. say "the projected increase in proportional risk is clearly insufficient to lead to the reestablishment of endemicity."

Childs et al. (2006) present a detailed analysis of malaria incidence in northern Thailand based on a quarter-century monthly time series (January 1977 through January 2002) of total malaria cases in the country's 13 Northern provinces. Over this time period, when IPCC claims the world warmed at a rate and to a level unprecedented over the prior one to two millennia, Childs et al. report there was an approximately constant rate of decline in total malaria incidence (from a mean monthly incidence in 1977 of 41.5 cases per hundred thousand people to 6.72 cases per hundred thousand people in 2001). Noting "there has been a steady reduction through time of total malaria incidence in northern Thailand, with an average decline of 6.45% per year," they say this result "reflects changing agronomic practices and patterns of immigration, as well as the success of interventions such as vector control programs, improved availability of treatment and changing drug policies."

Reiter (2008) came to similar conclusions, writing "simplistic reasoning on the future prevalence of malaria is ill-founded; malaria is not limited by climate in most temperate regions, nor in the tropics, and in nearly all cases, 'new' malaria at high altitudes is well below the maximum altitudinal limits for transmission." He further states, "Future changes in climate may alter the prevalence and incidence of the disease, but

obsessive emphasis on 'global warming' as a dominant parameter is indefensible; the principal determinants are linked to ecological and societal change, politics and economics."
Hulden and Hulden (2009) analyzed malaria statistics collected in Finland from 1750 to 2008 via correlation analyses between malaria frequency per million people and all variables that have been used in similar studies throughout other parts of Europe, including temperature data, animal husbandry, consolidation of land by redistribution, and household size. Over the entire period, "malaria frequency decreased from about 20,000–50,000 per 1,000,000 people to less than 1 per 1,000,000 people," they report. The two Finnish researchers conclude, "Indigenous malaria in Finland faded out evenly in the whole country during 200 years with limited or no counter measures or medication," making that situation "one of the very few opportunities where natural malaria dynamics can be studied in detail." Their study indicates "malaria in Finland basically was a sociological disease and that malaria trends were strongly linked to changes in the human household size and housing standard."

Effects of climate and socioeconomic factors on the projected future global distribution of malaria.

Source: Béguin et al. (2011).

The many findings described above make it clear a vast body of scientific examination and research contradict the claim that malaria will expand across the globe and intensify as a result of CO2-induced warming.

References

Béguin, A., Hales, S., Rocklöv, J., Åström, C., Louis, V.R., and Sauerborn, R. 2011. The opposing effects of climate change and socio-economic development on the global distribution of malaria. Global Environmental Change 21: 1209–1214.

Bosello, F., Roson, R., and Tol, R.S.J. 2006. Economy-wide estimates of the implications of climate change: human health. Ecological Economics 58: 579–591.

Breman, J.G. 2001. The ears of the hippopotamus: manifestations, determinants, and estimates of the malaria burden. American Journal of Tropical Medicine and Hygiene 64: 1–11.

Childs, D.Z., Cattadori, I.M., Suwonkerd, W., Prajakwong, S., and Boots, M. 2006. Spatiotemporal patterns of malaria incidence in northern Thailand. Transactions of the Royal Society of Tropical Medicine and Hygiene 100: 623–631.

Gething, P.W., Smith, D.L., Patil, A.P., Tatem, A.J., Snow, R.W., and Hay, S.I. 2010. Climate change and the global malaria recession. Nature 465: 342–345.

Githeko, A.K. and Ndegwa, W. 2001. Predicting malaria epidemics in the Kenyan highlands using climate data: A tool for decision makers. Global Change and Human Health 2: 54–63.

Hay, S.I., Cox, J., Rogers, D.J., Randolph, S.E., Stern, D.I., Shanks, G.D., Myers, M.F., and Snow, R.W. 2002. Climate change and the resurgence of malaria in the East African highlands. Nature 415: 905–909.

Hay, S.I., Rogers, D.J., Randolph, S.E., Stern, D.I., Cox, J., Shanks, G.D., and Snow, R.W. 2002. Hot topic or hot air? Climate change and malaria resurgence in East African highlands. Trends in Parasitology 18: 530–534.

Hulden, L. and Hulden, L. 2009. The decline of malaria in Finland—the impact of the vector and social variables. Malaria Journal 8: 10.1186/1475-2875-8-94.

Jackson, M.C., Johansen, L., Furlong, C., Colson, A., and Sellers, K.F. 2010. Modelling the effect of climate change on prevalence of malaria in western Africa. Statistica Neerlandica 64: 388–400.

Kuhn, K.G., Campbell-Lendrum, D.H., Armstrong, B., and Davies, C.R. 2003. Malaria in Britain: Past, present, and future. Proceedings of the National Academy of Science, USA 100: 9997–10001.

Lieshout, M.V., Kovats, R.S., Livermore, M.T.J., and Martens, P. 2004. Climate change and malaria: analysis of the SRES climate and socio-economic scenarios. Global Environmental Change 14: 87–99.

Nabi, S.A. and Qader, S.S. 2009. Is global warming likely to cause an increased incidence of malaria? Libyan Journal of Medicine 4: 18–22.

Nkurunziza, H. and Pilz, J. 2011. Impact of increased temperature on malaria transmission in Burundi. International Journal of Global Warming 3: 77–87.

Paaijmans, K.P., Blanford, S., Chan, B.H.K., and Thomas, M.B. 2012. Warmer temperatures reduce the vectorial capacity of malaria mosquitoes. Biology Letters 8: 465–468.

Reiter, P. 2001. Climate change and mosquito-borne disease. Environmental Health Perspectives 109: 141–161.

Reiter, P. 2000. From Shakespeare to Defoe: Malaria in England in the Little Ice Age. Emerging Infectious Diseases 6: 1–11.

Reiter, P. 2008. Global warming and malaria: knowing the horse before hitching the cart. Malaria Journal 7 (Supplement 1): 10.1186/1475-2875-7-S1-S3.

Reiter, P., Lathrop, S., Bunning, M., Biggerstaff, B., Singer, D., Tiwari, T., Baber, L., Amador, M., Thirion, J., Hayes, J., Seca, C., Mendez, J., Ramirez, B., Robinson, J., Rawlings, J., Vorndam, V., Waterman, S., Gubier, D., Clark, G., and Hayes, E. 2003. Texas lifestyle limits transmission of Dengue virus. Emerging Infectious Diseases 9: 86–89.

Rogers, D.J. and Randolph, S.E. 2006. Climate change and vector-borne diseases. Advances in Parasitology 62: 345–381.

Russell, R.C. 2009. Mosquito-borne disease and climate change in Australia: time for a reality check. Australian Journal of Entomology 48: 1–7.

Shanks, G.D., Biomndo, K., Hay, S.I., and Snow, R.W. 2000. Changing patterns of clinical malaria since 1965 among a tea estate population located in the Kenyan highlands. Transactions of the Royal Society of Tropical Medicine and Hygiene 94: 253–255.

Shanks, G.D., Hay, S.I., Stern, D.I., Biomndo, K., and Snow, R.W. 2002. Meteorologic influences on Plasmodium falciparum malaria in the highland tea estates of Kericho, Western Kenya. Emerging Infectious Diseases 8: 1404–1408.

Small, J., Goetz, S.J., and Hay, S.I. 2003. Climatic suitability for malaria transmission in Africa, 1911–1995. Proceedings of the National Academy of Sciences USA 100: 15,341–15,345.

Stern, D.I., Gething, P.W., Kabaria, C.W., Temperley, T.H., Noor, A.M., Okiro, E.A., Shanks, G.D., Snow, R.W., and Hay, S.I. 2011. Temperature and Malaria Trends in Highland East Africa. PLoS One 6: 10.1371/ journal.pone.0024524.

Sun, K.N., Kain, K.C., and Keystone, J.S. 2004. Malaria. Canadian Medical Association Journal 170: 1693–1702.

Thomas, C. 2004. Malaria: a changed climate in Africa? Nature 427: 690–691.

Tol, R.S.J. and Dowlatabadi, H. 2001. Vector-borne diseases, development & climate change. Integrated Assessment 2: 173–181.

Tuchman, N.C., Wahtera, K.A., Wetzel, R.G., Russo, N.M., Kilbane, G.M., Sasso, L.M., and Teeri, J.A. 2003. Nutritional quality of leaf detritus altered by elevated atmospheric CO2: effects on development of mosquito larvae. Freshwater Biology 48: 1432–1439.

WHO, WMO, UNEP. 2003. Climate Change and Human Health—Risks and Responses: Summary. Geneva, Switzerland.

Zhou, G., Minakawa, N., Githeko, A.K., and Yan, G. 2004. Association between climate variability and malaria epidemics in the East African highlands. Proceedings of the National Academy of Sciences, USA 101: 2375–2380.

Dengue Fever

Concerns over large increases in dengue fever as a result of rising temperatures are unfounded and unsupported by the scientific literature, as climatic indices are poor predictors for dengue fever.

According to Ooi and Gubler (2009), "dengue/dengue hemorrhagic fever is the most important vector-borne viral disease globally," with more than half the world's population living in areas deemed to be at risk of infection. Kyle and Harris (2008) note "dengue is a spectrum of disease caused by four serotypes of the most

prevalent arthropod-borne virus affecting humans today," and "its incidence has increased dramatically in the past 50 years," to where "tens of millions of cases of dengue fever are estimated to occur annually, including up to 500,000 cases of the life- threatening dengue hemorrhagic fever/dengue shock syndrome." Some of the research papers summarized in previous sections address dengue fever as well as malaria. With a few worthy exceptions, we do not repeat those summaries in this section. The most important exceptions are papers written by or coauthored by Paul Reiter (2001, 2003, 2010a, 2010b), one of the world's premier authorities on the subject. Reiter analyzed the history of malaria and dengue fever in an attempt to determine whether the incidence and range of influence of these diseases would indeed increase in response to CO2-induced global warming.

His reviews established what is now widely accepted among experts in the field, that the natural history of these vector-borne diseases is highly complex, and the interplay of climate, ecology, vector biology, and a number of other factors defy definition by the simplistic analyses utilized in the computer models relied on by environmental activists and the IPCC.

That there has in fact been a resurgence of these diseases in parts of the world is true, but as Reiter (2001) notes; it is "facile to attribute this resurgence to climate change." This he shows via a number of independent analyses that clearly demonstrate factors associated with politics, economics, and human activity is the principal determinants of the spread of these diseases. He describes these factors as being "much more significant" than climate in promoting disease expansion. Two years later, Reiter took up the subject again, this time with 19 other scientists as coauthors (Reiter et al., 2003), and yet again in 2010. Reiter's work remains the most comprehensive critique of the claims of the Intergovernmental Panel on Climate Change.

Kyle and Harris (2008) wrote "there has been a great deal of debate on the implications of global warming for human health," but "at the moment, there is no consensus." However, "in the case of dengue," they report, "it is important to note that even if global warming does not cause the mosquito vectors to expand their geographic range, there could still be a significant impact on transmission in endemic regions," because "a 2°C increase in temperature would simultaneously lengthen the lifespan of the mosquito and shorten the extrinsic incubation period of the dengue virus, resulting in more infected mosquitoes for a longer period of time." Nevertheless, they state there are "infrastructure and socioeconomic differences that exist today and already prevent the transmission of vector-borne diseases, including dengue, even in the continued presence of their vectors," citing Reiter (2001).

Wilder-Smith and Gubler (2008) conducted a review of the scientific literature, noting "the past two decades saw an unprecedented geographic expansion of dengue" and "global climate change is commonly blamed for the resurgence of dengue," but they add, "There are no good scientific data to support this conclusion." The two researchers report, "Climate has rarely been the principal determinant of [their] prevalence or range," and "human activities and their impact on local ecology have generally been much more significant." They cite as contributing factors "urbanization, deforestation, new dams and irrigation systems, poor housing, sewage and waste management systems, and lack of reliable water systems that make it necessary to collect and store water," further noting "disruption of vector control programs, be it for reasons of political and social unrest or scientific reservations about the safety of DDT, has contributed to the resurgence of dengue around the world."

In addition, Wilder-Smith and Guble write "large populations in which viruses circulate may also allow more co-infection of mosquitoes and humans with more than one serotype of virus," which would appear to be borne out by the fact that "the number of dengue lineages has been increasing roughly in parallel with the size of the human population over the last two centuries." Most important, perhaps, is "the impact of international travel," of which they say "humans, whether troops, migrant workers, tourists, business travelers, refugees, or others, carry the virus into new geographic areas," and these movements "can lead to

epidemic waves." The two researchers conclude, "Population dynamics and viral evolution offer the most parsimonious explanation for the observed epidemic cycles of the disease, far more than climatic factors." Russell et al. (2009) showed the dengue vector (the Aedes Aegypti mosquito) "was previously common in parts of Queensland, the Northern Territory, Western Australia and New South Wales," and it had, "in the past, covered most of the climatic range theoretically available to it," adding "the distribution of local dengue transmission has [historically] nearly matched the geographic limits of the vector." This being the case, they conclude the vector's current absence from much of Australia "is not because of a lack of a favorable climate." Thus, they reason "a temperature rise of a few degrees is not alone likely to be responsible for substantial increases in the southern distribution of A. Aegypti or dengue, as has been recently proposed." Instead of futile attempts to limit dengue transmission by controlling the world's climate, therefore, the medical researchers recommend "well resourced and functioning surveillance programs, and effective public health intervention capabilities, are essential to counter threats from dengue and other mosquito-borne diseases."

Reiter (2010a) observed "the introduction and rapidly expanding range of Aedes Albopictus in Europe is an iconic example of the growing risk of the globalization of vectors and vector-borne diseases," and "the history of yellow fever and dengue in temperate regions confirms that transmission of both diseases could recur, particularly if Aedes Aegypti, a more effective vector, were to be re-introduced." He states "conditions are already suitable for transmission." Much more important than a rise or fall of a couple degrees of temperature, Reiter says, is "the quantum leap in the mobility of vectors and pathogens that has taken place in the past four decades, a direct result of the revolution of transport technologies and global travel."

Carbajo et al. (2012) evaluated the relative contributions of geographic, demographic, and climatic variables to the recent spread of dengue in Argentina. They found dengue spatial occurrence "was positively associated with days of possible transmission, human population number, population fall and distance to water bodies." When considered separately, the researchers write, "the classification performance of demographic variables was higher than that of climatic and geographic variables." Thus, although useful in estimating annual transmission risk, Carbajo et al. conclude temperature "does not fully describe the distribution of dengue occurrence at the country scale," and "when taken separately, climatic variables performed worse than geographic or demographic variables."

These several observations indicate concerns over large increases in dengue fever as a result of rising temperatures are unfounded and unsupported by the scientific literature, as climatic indices are poor predictors for dengue fever.

References

Carbajo, A.E., Cardo, M.V., and Vezzani, D. 2012. Is temperature the main cause of dengue rise in non-endemic countries? The case of Argentina. International Journal of Health Geographics 11: 10.1186/1476-072X-11-26.

Johansson, M.A., Cummings, D.A.T., and Glass, G.E. 2009. Multiyear climate variability and dengue-El Niño Southern Oscillation, weather and dengue incidence in Puerto Rico, Mexico, and Thailand: A longitudinal data analysis. PLoS Medicine 6: e1000168.

Kyle, J.L. and Harris, E. 2008. Global spread and persistence of dengue. Annual Review of Microbiology 62: 71–92.

Ooi, E.-E. and Gubler, D.J. 2009. Global spread of epidemic dengue: the influence of environmental change. Future Virology 4: 571–580.

Reiter, P. 2001. Climate change and mosquito-borne disease. Environmental Health Perspectives 109: 141–161.

Reiter, P. 2010a. Yellow fever and dengue: A threat to Europe? Eurosurveillance 15:eurosurveillance.org/ViewArticle.aspx?Articleid=19509.

Reiter, P. 2010b. A mollusc on the leg of a beetle: Human activities and the global dispersal of vectors and vector-borne pathogens. In: Relman, D.A., Choffnes, E.R., and Mack, A. (Rapporteurs). Infectious Disease Movement in a Borderless World. The National Academies Press, Washington, DC, USA, p. 150–165.

Reiter, P., Lathrop, S., Bunning, M., Biggerstaff, B., Singer, D., Tiwari, T., Baber, L., Amador, M., Thirion, J., Hayes, J., Seca, C., Mendez, J., Ramirez, B., Robinson, J., Rawlings, J., Vorndam, V., Waterman, S., Gubier, D., Clark, G., and Hayes, E. 2003. Texas lifestyle limits transmission of Dengue virus. Emerging Infectious Diseases 9: 86–89.

Rohani, P. 2009. The link between dengue incidence and El Niño Southern Oscillation. PLoS Medicine 6: e1000185.

Russell, R.C. 2009. Mosquito-borne disease and climate change in Australia: time for a reality check. Australian Journal of Entomology 48: 1–7.

Russell, R.C., Currie, B.J., Lindsay, M.D., Mackenzie, J.S., Ritchie, S.A., and Whelan, P.I. 2009. Dengue and climate change in Australia: predictions for the future should incorporate knowledge from the past. Medical Journal of Australia 190: 265–268.

Shang, C.-S., Fang, C.-T., Liu, C.-M., Wen, T.-H., Tsai, K.-H., and King, C.-C. 2010. The role of imported cases and favorable meteorological conditions in the onset of dengue epidemics. PLoS 4: e775.

Wilder-Smith, A. and Gubler, D.J. 2008. Geographic expansion of Dengue: The impact of international travel. Medical Clinics of North America 92: 1377–1390.

Tick-borne Diseases

Climate change has not been the most significant factor driving the recent temporal patterns in the epidemiology of tick-borne diseases.

Sarah Randolph of the University of Oxford's Department of Zoology is a leading scholar on tick-borne diseases. She and fellow Oxford faculty member David Rogers observed in 2000 that tick-borne encephalitis (TBE) "is the most significant vector-borne disease in Europe and Eurasia," having "a case morbidity rate of 10–30% and a case mortality rate of typically 1–2% but as high as 24% in the Far East." The disease is

caused by a flavivirus (TBEV), which is maintained in natural rodent-tick cycles; humans may be infected with it if bitten by an infected tick or by drinking untreated milk from infected sheep or goats.

Early discussions on the relationship of TBE to global warming predicted the disease would expand its range and become more of a threat to humans in a warmer world. However, Randolph and Rogers (2000) note, "like many vector-borne pathogen cycles that depend on the interaction of so many biotic agents with each other and with their abiotic environment, enzootic cycles of TBEV have an inherent fragility," so "their continuing survival or expansion cannot be predicted from simple univariate correlations."

Randolph (2010) examined the roles played by various factors that may influence the spread of tick-borne diseases. After describing some of the outbreaks of tick-borne disease in Europe over the past couple of decades, Randolph states "the inescapable conclusion is that the observed climate change alone cannot explain the full heterogeneity in the epidemiological change, either within the Baltic States or amongst Central and Eastern European countries," citing Sumilo et al. (2007). Instead, she writes, "a nexus of interrelated causal factors—abiotic, biotic and human—has been identified," and "each factor appears to operate synergistically, but with differential force in space and time, which would inevitably generate the observed epidemiological heterogeneity."

Many of these factors, she continues, "were the unintended consequences of the fall of Soviet rule and the subsequent socio-economic transition (Sumilo et al., 2008b)," among which she cites "agricultural reforms resulting in changed land cover and land use, and an increased reliance on subsistence farming; reduction in the use of pesticides, and also in the emission of atmospheric pollution as industries collapsed; increased unemployment and poverty, but also wealth and leisure time in other sectors of the population as market forces took hold."

Randolph concludes "there is increasing evidence from detailed analyses that rapid changes in the incidence of tick-borne diseases are driven as much, if not more, by human behavior that determines exposure to infected ticks than by tick population biology that determines the abundance of infected ticks," as per Sumilo et al. (2008a) and Randolph et al. (2008). She ends her analysis by stating, "While nobody would deny the sensitivity of ticks and tick-borne disease systems to climatic factors that largely determine their geographical distributions, the evidence is that climate change has not been the most significant factor driving
the recent temporal patterns in the epidemiology of tick-borne diseases."

References

Estrada-Peña, A. 2003. Climate change decreases habitat suitability for some tick species (Acari: Ixodidae) in South Africa. Onderstepoort Journal of Veterinary Research 70: 79–93.

Randolph, S.E. 2001. Tick-borne encephalitis in Europe. The Lancet 358: 1731–1732.

Randolph, S.E. 2010. To what extent has climate change contributed to the recent epidemiology of tick-borne diseases? Veterinary Parasitology 167: 92–94.

Randolph, S.E., Asokliene, L., Avsic-Zupanc, T., Bormane, A., Burri, C., Golovljova, I., Hubalek, Z., Knap, N., Kondrusik, M., Kupca, A., Pejcoch, M., Vasilenko, V., and Zygutiene,
M. 2008. Variable spikes in TBE incidence in 2006 independent of variable tick abundance but related to weather. Parasites and Vectors 1: e44.

Randolph, S.E. and Rogers, D.J. 2000. Fragile transmission cycles of tick-borne encephalitis virus may be disrupted by predicted climate change. Proceedings of the Royal Society of London Series B 267: 1741–1744

Sumilo, D., Asokliene, L., Avsic-Zupanc, T., Bormane, A., Vasilenko, V., Lucenko, I., Golovljova, I., and Randolph, S.E. 2008a. Behavioral responses to perceived risk of tick-borne encephalitis: vaccination and avoidance in the Baltics and Slovenia. Vaccine 26: 2580–2588.

Sumilo, D., Asokliene, L., Bormane, A., Vasilenko, V., Golovljova, I., and Randolph, S.E. 2007. Climate change cannot explain the upsurge of tick-borne encephalitis in the Baltics. PLos ONE 2: e500.

Sumilo, D., Bormane, A., Asokliene, L., Vasilenko, V., Golovljova, I., Avsic-Zupanc, T., Hubalek, Z., and Randolph, S.E. 2008b. Socio-economic factors in the differential upsurge of tick-borne encephalitis in Central and Eastern Europe. Reviews in Medical Virology 18: 81–95.

Conclusion

IPCC fails to acknowledge the human health benefits of a warming world, claiming instead that the net effect of warming is a cost rather than a benefit.

Fossil fuels have benefited human health by making possible the dramatic increase in human prosperity since the first Industrial Revolution, making investments possible in goods and services that are essential to protecting human health and prolonging human life. Fossil fuels further improve human health by making environmental protection both valued and financially possible and by powering technologies and production of goods and services, transportation, communication that all improve quality of life, and protect human health and welfare, extend life spans.
If the combustion of fossil fuels leads to some amount of global warming, then the positive as well as negative health effects of that warming should be included in any cost-benefit analysis of fossil fuels.
Medical science explains why colder temperatures often cause diseases and sometimes fatalities whereas warmer temperatures are associated with health benefits.
Empirical research confirms that warmer temperatures lead to a net decrease in temperature- related mortality in virtually all parts of the world, even those with tropical climates. The evidence of this benefit comes from research conducted in nearly every major country of the world.
Global warming is reducing the incidence of fatal coronary events related to low temperatures and wintry weather by a much greater degree than it increases the incidence of heat related illness or death attributable to heat waves. Respiratory illness, strokes and myocardial infarction are less frequent during unseasonably warm periods than during unseasonably cold periods.
Global warming is reducing the incidence of death due to respiratory disease in many parts of the world, including Spain, Canada, Shanghai, and even on the subtropical island of Taiwan. Low minimum temperatures have been found to be a stronger risk factor than high temperatures for outpatient visits for respiratory diseases. Warm weather reduces the incidence of death due to stroke around the world.
A vast body of scientific examination and research contradicts and refutes the claim that malaria will expand across the globe or intensify in some regions as a result of any predicted CO2-induced warming. Concerns over large increases in mosquito-transmitted dengue fever as a result of rising temperatures are unfounded and unsupported by the scientific literature.

While climatic factors largely determine the geographical distribution of ticks, temperature and climate change are not among the significant factors determining the incidence of tick-borne diseases.
In the face of this extensive evidence of the positive effects of fossil fuels on human health, IPCC continues to claim the net impact on human health of fossil fuels will be negative. Because virtually all cost-benefit analyses incorporate the IPCC's incorrect assumptions into their calculation of the social cost of fossil fuels, they are unreliable guides to policymakers.

12. Conclusion

This is not a complete expose of the misconduct of the US EPA sponsored researchers and in house science and policy staff in matters of epidemiology and toxicology and it focuses on the US EPA research/ policy /regulatory activities in air quality science and policy making—an equally scandalous case can be made for US EPA work in other areas of responsibility where toxicology and epidemiology are abused and misused to expand the EPA list of targets for regulation and opportunities for EPA to scaremonger.

I also cannot take the time or the space in this discussion to expose the US EPA new area of scientific misconduct and scaremongering—epigenetics and their claims of inheritable acquired toxin carcinogenic genetic mutations—revisiting Lamarck and Lysenko long ago discredited theories about acquired genetic changes. Such irresponsible scares about inherited toxic and cancer effects are ideal for irresponsible aggressive environmental fanatic wannabee regulators and their obedient research army.

US EPA researchers in epigenetics are their new breed of scaremongers with the target people who think exposure to some named toxin might effect their children or grandchildren. The lust of power and influence and cheating on science go hand in hand.

All of what I have exposed above combines to make an effective and urgent argument for the proposed US EPA policy change to promote integrity and transparency of US EPA science in matters of regulatory policy decision making. The time for cleaning up the US EPA scientific perfidy and misconduct, malfeasance is long past overdue.

I anticipate there will be institutions and scientists panicked and anxious about proving up their research assertions and conclusions—a very beneficial and healthy development. The polity will benefit from science and policy making that is based on reliable methods used by researchers with integrity who are subjected to impartial and thorough competent reviews by experts who are not conflicted by ideological, political, monetary or social/professional influences.

John Dale Dunn MD JD

Personal Matters / Ex. 6